CERTIFICATE

This is to certify that the dissertation entitled "**VENPULLI"** is a bonafide work done by **Dr. R. NANDHINI**, Government Siddha Medical College, Chennai – 600 106 in partial fulfilment of the University rules and regulations for award of **SIDDHA MARUTHUVA PERARIGNAR** under my guidance and supervision during the academic year 2013 – 2016.

Name & Signature of the Guide

Name & Signature of the Head of Department

Name & Signature of the Dean/ Principal

AN OPEN CLINICAL STUDY ON VENPULLI (VITILIGO) WITH THE EVALUATION OF SIDDHA DRUG RASACHEENEE CHOORANAM (INTERNAL) & KARKADAGASINGI PATTRU (EXTERNAL)

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INTRODUCTION

INTORDUCTION

நந்தீசர் மூலரகத் தியருஞ் சட்டை
நாதரொடு பதஞ்சலி வியாக்கிரம பாதர்
சுந்தரானந்தர் மச்சமுனி புண்ணாக்கீசர்
சுருதிகண்ட கமலமுனி கொங்கணரும் போகர்
மந்திரஞ்சேர் கோரக்கர் சண்டிகேசர்
வரராமர் காலாங்கியார் கூனக்கண்ணர்
தந்திரருஞ்சேர் கருவூரார் ரிஷிகள் தேவர்
தபோதனர்கள் பாதமா தார மாமே[1]

The siddha science is a traditional treatment system generated from Dravidian culture[2]. The concept of system is the holistic system of codified life style a health care perfected many thousands of years .The siddha that system has a healthy soul can only be developed through healthy body,so they developed methods and medication that are believed to strengthen their physical body and thereby their soul. The siddha were many year ago in the Tamilnadu.This system is also called Tamil Maruthuvam[3]. Siddha focused to "Ashtamasiddhi" the eight super natural power.Those who attained or achieved the above said powers are known as siddhar .There were eighteen important siddhars in olden days and they developed this system of medicine.Hence it is called siddha medicine.[4]Generally the basic concepts of the siddha medicine recognizes predominance of vaadham, piththam,and kabham in childhood ,adulthood,and old age respectively[5]. The treatment of siddha medicine is aiming to keep the three humors in equilibrium and maintenance of seven elements .The treatment should be commenced as early as possible after assessing the course and cause of the disease.

The drug use in siddha medicines were based five elements and treatment of five properties:taste(suvai),character(gunam),potency(veeryam),class(pirivu),action(magiami)

According to the mode of application ,the siddha medicine could be categorized into two classes ;Internal medicine and external medicine[6].Siddhar were classified the diseases on the basis of affected dhosam ,affected kosam into 4448 diseases. Among that yugi was classified the kuttam as 18. Venpulli is one among them.[7]

In modern science venpulli is termed as Vitiligo is a condition in which the pigment will be lost from the particular areas of the skin causing whitish patches, often with no clear cause .A condition in which the skin turns white due to the loss of pigment melanin that gives the skin[8]. Siddha system clearly lays down the general principles of body constituents in the classic bootha system .They hold that the universe is a macrocosum made up of the five primordial elements or boothas ,viz nilam(earth),neer (water),thee(fire),vali(wind),veli (space) and the human being is a microcosum made up of these five element. When there is a change in equilibirium of this five elements in macro cosum it influence the equilibirium of five elements in micro cosum.This imbalance leads to the course of a disease.[9]

In the present days the environment is highly disturbed .The air is polluted, water is contaminated and food is mostly adulterated. These changes cause several disease in human beings .one of the disease the human are suffering is venpulli in modern medicine is related to vitiligo or leucoderma. The intention of this study of the author is to educate help and treat the patient suffering from venpulli .There are many challenging medicine found in siddha literature which play a miracle for treating skin disease especially vitiligo.vitiligo isa chronic skin condition characterized by portions of the skin losing their pigment[10].The global percentage of people affected with vitiligo is less than 1% with some populations averaging 2-3% and rarely as high as 16% .The disease shows no regard to the ethnic, racial, or socio economic background of the affected sufferers. The cosmetic impact of this disease is tremendous and its psychological impact devastating particularly in coloured races[11].Among them the author is interested to prove the efficacy of trial drugs.In the treatment of skin disease the Siddha system having wonderful medicine. So the author selected venpulli as her dissertation topic for which I have chosen.

Rasacheenee chooranam –Internal[12]

Karkadagasingi pattru –External [13]

AIM AND OBJECTIVE

AIM AND OBJECTIVES

AIM:

The aim of my study is to evaluate the efficacy and safety of the siddha drug **RASACHEENEE CHOORANAM (internal) and KARKADAGASINGI PATTRU (EXTERNAL)** both clinically and experimentally in the treatment of **VENPULLI.**

OBJECTIVES:

- To collect the authorial measures and literature reviews of **VENPULLI** in ancient siddha and modern literatures.
- Have occupationan idea of the incidence of the disease with regard to age, sex, precipitating, socio economic status, food, kaalam and factors etc.
- To expose the efficacy of siddhar's diagnostic principles.
- To utilize the modern investigation methods to confirm the diagnosis and prognosis.
    - ✓ To have clinical trial on patients with **VENPULLI** with selected siddha medicine. **RASACHEENEE CHOORANAM (INTERNAL) and KARKADAGASINGI PATTRU (EXTERNAL)**
- To evaluate
    - ✓ Toxicological screening
        - Acute
        - Sub acute
        - Sub chronic
- To find out the statistical analysis of clinical study
    - Biochemical analysis
    - Physico chemical analysis
    - Bio-Statistical analysis

REVIEW OF LITERATURE

SIDDHA ASPECT

SIDDHA ASPECT

இயல்:

தோல் தடிப்புண்டாகி உடம்பு முழுதும் தவளம்போல வெளுப்பாகும்.

இதில் மயிர் வெளுப்பானால் அசாத்தியமாகும்.உதடு,உள்ளங்கை.குதம்,

குய்யம்,நெருப்புபட்ட புண் போல் நிறமிருந்தால் அசாத்தியம்,மேனி

வெடிப்பாகி வெளுத்து வீங்கும்[14].

Venpulli (in yugi's classification it is under 18 types or kuttam as swetha kuttam) Swetha kuttam (swetha –white)

WHEN TO SUSPECT /RECOGNISE (DIAGNOSTIC CRITERIA)

Venpulli is a chronic skin disease characterized by various sizes of hypopigmentar patches in the skin.According to siddha literatures if the lesions may be present in palms,soles, anal region, genetalia are incurable.

தடிப்பாகத் தவளனிரம் போல் வெளுத்துச்
சர்வாங்கமும் வெளுத்தாற்றான் றீரும்பும்
மடிப்பாக மயிர்வெளுத்தா லசாத்ய மாகும்
வரிவுதடுவுள் ளங்கைக்குதங்குய்யந்
நெடிப்பாக நெருப்புப்பட்டது போற்புண்ணாய்
நிறமிருந்தா லசாத்தியமென்றே யுரைக்க்கலாகும
வெடிப்பாக மேனியெல்லாம் வெளுத்து வீங்கில்
வெண்சுவேத குட்டமென்றே விளம்ப லாமே.[15]

Aetiology:

According to siddha system, the predisposing causes for this disease have been described as hereditary factor, stress, and strain, malnutrition and veneral exposure and no other specific causes have been mentioned for venpulli.

According to Thirumoolar karukkidaivaithiya nool

வியாதியுண் மூவாறு விலங்கிய குட்டங்கேள்
சுயாதிக் கிரந்தி சுழன் மேகத்தாலாரும்
பயாதி மண்ணுளப் பலவண்டினா லெட்டும்
நியாதி புழுனாலாய் நின்றதிக் குட்டமே[16]

Six types of kuttam i.e skin disease are caused by kirandhi and megham, eight types are caused by insects in the soil, and four types are caused by worms.

ACCORDING TO YUGHI VAITHIYA CHINTHAMANI 800

விளம்பவே மிகுந்த உஷ்ணந்தன் னாலும்
மிகுந்த சீதளத்தாலு மழற்சி யாலும்
விளம்பவே மந்தத்தால் வாந்தி யாலும்
மகத்தான பெண்ணோடு மருவலலாலும்
கிளம்பவே கிலேசங்கள் மிகுதலாலும்
கெடியான வுறக்கங்கள டைத லாலும்
தளம்பவே மயிருகற்கள் தவிடு மண்கள்
சாதத்திற் பருகலால் மிகுக்குங் குஷ்டம்.

Excessive heat and cold, vomiting due to indigestion, unbridled sexual indulgence,and excessive sleep in day time, frequent intake of food mixed with stone and hair.

குட்டந்தான் பதினெட்டு வரவே தென்னிற்
குருனிந்தை சிவனிந்தை மரையோர் நிந்தை
திட்டந்தான் தேவதையைத் தூஷனைக்கு ரோதம்
செப்பலாற் றிருடலாற் பரதா ரத்தை
அட்டந்தா னாசையால டைக்க லத்தை
அபகரித்த லகதிபர தேசி தன்னை
வட்டந்தான் வைதலார் கற்பழித்தல்
வந்திடுமே பதினெட்டு குட்டந்தான்.

Use of indescent and disrespectful words against god and highly religious and noble people, neglecting phans and beggars, intention to spoil others, raping, greed, cursing the elders and so on have also been given as predisposing causes by yughi text.These habits are supposed to be the factors which lower the immunity of the body (iyarkai vanmai) and make it vulnerable to the disease.[17]

ACCORDING TO AGASTHIYAR KANMA KAANDAM:

சேர்ந்தே குட்டமொடு குறைநோய்கள் வந்த
சேதி கேள் மலராத வரும்பு கொய்தல்
தாரிந் சீவசெந்து வதைகள் செய்தல்
தாய்தந்தை மனது நொந்து ரோந் தானே
தானென்ற தெய்வரு தனையழித்தல்
சார்வான பெரியோர்கள் தமைப் பழித்தல்
கானென்ற நந்தவனம் பூஞ்செடிகள் வெட்டல்
கருமமடா சரீ ரத்திற் காசுபோலே

ஊனென்ற வுடம்பெல்லாம் பொட்டு பொட்டா
யுடன் வெளூத்து குறைநோயுதிரஞ் சிந்தும்
வானென்ற கருமங்கள் தீர்ப்பதற்கு
வகையொன்று சொல்வேன்கேள்.

மலராத அரும்புகொய்தல் சீவசெந்துகளை வதைகள்செய்தல், பெற்றோர் மனதை நோகும்படி செய்தல் பெரியோர்கள் தம்மைபழித்தல்,நந்தவனம் பூச்செடிகள் வெட்டுதல் ஆகிய இக்காரணங்களால் குட்டநோய் வரும் என்று அகத்தியர் கன்ம காண்டத்தில் கூறப்பட்டுள்ளது.[18]

CLASSIFICATION:

ACCORDING TO YUGI VAITHIYA CHINTHAMANI:

“முத்தாகும் குட்ட்ந்தான் பதினெட்டுக்கும்
முனியான யூகினான் சொல்லக் கேளாய்
புத்தாகும் புண்டரீக குஷ்டத்தோடு
பெருகின்ற விற்போடகக் குட்டமாகும்
பத்தாகும் பாமா குஷ்ட ஏகசர்ம குஷ்டம்
பரிவான கர்னகுஷ்டம் சர்மகுஷ்டம்
கித்தாகுங் கிருஷ்ண குட்டம் அவுதும்பர குட்டம்
கேடியான மண்டல குஷ்டமாகு மென்னே
குட்டமா மபரிச குஷ்ட மோடு
மருவலாங் கிடிப குஷ்டந் சர்மதல குஷ்டந்
திட்டமாற் தத்துரு குஷ்ட மோடு
தக்கான சித்துமா குஷ்டஞ் சதாரு குஷ்டந்
துட்டமாஞ் சுவேத குஷ்டதன் னோடொக்கச்
சுயம்பான பதினெட்டுக் குட்டமாச்சே.

1.Pundareeka kuttam
2. Virpodaka kuttam
3. Baama kuttam
4. Gaja saruma kuttam
5. Karna kuttam
6. Siguram kuttam
7. Krishna kuttam
8. Avudhubaram
9. Mandala kuttam
10. Abarisa kuttam
11. Visarchika kuttam

12. Vibaathika kuttam

13.Kideeba kuttam

14. Sarmathala kuttam

15. Thethru kuttam

16. Sithuma kuttam

17. Sathaaru kuttam

18. Swetha kuttam[19]

ACCORDING TO SIDDHAR ARUVAI MARUTHUVAM

Venpadai has been classified into 3 types on the basis of mukkutram, they are,

1. Vaatha venpadai
2. Piththa venpadai
3. Kaba venpadai[20]

ACCORDING TO SIDDHA SIRAPPU MARUTHUVAM

Venpulli has been classified into 4 types:

1. Vaatha venpadai
2. Piththa venpadai
3. Kabha venpadai
4. Mega venpadai[21]

ACCORDING TO ATHMA RAKSHAMIRTHA VAIDHYA SARASANKIRAHAM

Venpadai is classified into 4 types

1. Venkuttam
2. Senkuttam
3. Karunkuttam
4. Peru viyathi[22]

CLINICAL FEATURES OF KUTTA ROGAM

1.ACCORDING TO THANVANTHIRI VAITHIYAM

"மீக்கௌத் தோறூமெலுமோர் முகம் வெளுக்குமாகில்

நோக்கியல் மரிக்குஞ் சொன்ன வெண்குட்டமாமே"[23].

2.ACCORDING TO VAITHIYA SAARASANGIRAHAM

Sole,Hands,lips,scalp,fingers and wrist joint-all those organs are found with white coloured patches which are circumscribed along with thickened border and gradually spread which is known as "venpadai".Blood, Muscles, and adipose tissue are affected by disease.

Discolouration of hairs ,absence of normal skin texture comparing the adjoining normal skin area and appearance of burns is not curable[24].

3. ACCORDING TO PARARAASA SEKARAM

1.Watery discharge

2. Grey colour

3. Foul smelling[25]

4. ACCORDING TO ANUBHAVA VAITHIYA DEVA RAGASIYAM

இந்நோயை குஷ்டமென கூறீனும் இது குஷ்ட வகைகளின்று வேறுபட்டது என்பதையும் குஷ்டத்தைப்போல் அவ்வளவு கொடுமையான வியாதி அல்லவென்றும் உணரவேண்டும்.இந்நோயில் திட்டுதிட்டாக வெண்மை நிறமான படைகள் உண்டாகி பிறகு தேகம் முழுவதும் பரவி உடலை விகாரப்படுத்ததுதல் முதலிய குணங்களை உடையது.

THREE TYPES

1.Vatha venpadai

2. Piththa venpadai

3. Kaba venpadai

CLINICAL FEATURES

1.The skin appears glittering and rough

2. There is an excessive perspiration

3. Discolouration
4. Heat and itching of the skin
5. Numbness in some parts of the body[26]

5. ACCORDING TO SIRAPPU MARUTHUVAM

1. Vaatha venpadai
2. Piththa venpadai
3. Kaba venpadai
4. Mega venpadai

1. VAATHA VENPADAI

It is characterized by the depigmented patches, which are dry, rough,reddish with somewhat pale black in colour.

2.PITHTHA VENPADAI

It is characterized by the depigmented patches red in colour like lotus flower ,spreading with burning sensation and loss of hairs on that area

3.KABA VENPADAI

Itis characterized by the depigmented patches white in colour like leucus flower spreads with rashes and itching

4.MEGA VENPADAI

It is due to the veneral disease and it occurs after 4 or 6 months after the onset as disease, syphilis within four or six months of the attack .this venpadai develops initially along the nape and the adjoining spaces .Also gradually it affects the shoulder joints, back of trunk. Clinical features of this type are clearly defined by the author of “siddha maruthuva sirappu “as follow as depigmented patches are small in number .pale in colour ,turmeric colour or dark colour margin marked with hyperpigmented signs .These lesion are circumscribed with 2mm to 3mm diameter or above .this correct picture of

hypopigmented and hyper pigmented skin seems to be more or less a multi eyed filter (sieve –like)

Females are more prone to this mega venpadai, therefore anti –syphilitic theraphy is mandatory in the early period of the treatment .

CHARACTER OF VENPULLI

Skin color will change to reddish black or reddish white or white colour with spreading nature .the imbalance of the three thathus produces certain lesions in skin known as kuttam.

Absence of perspiration and thickening of skin may produce the colour changes in skin

தீரும் ,தீராதவை

சாத்தியம்-11

பூண்டந் நுரவினோடு சதாரிகம் புண்டரீ கந்த

தாண்டு விற்போடம் பாமாவுடன் மைதலம் வெங்குட்டம்

கூண்டிடு காகறந்தி சிறுமை யசல குட்டம்

வேண்டிய வியாதியோடும் பதினொன்றாம் விரித்துக் கானே.

அசாத்தியம்

சொல்லுகுட்டம் எழுவகைபேர் சொல்லிக் கபால சர்மீகம்

வெல்லு முதும்பா மேகிடிபம் விசர்ச்சிமண்டலக் கிரமும்

மல்லல் தருமீசி யகுவை யாகும் பெயரோ ரேழாகும

வல்லகியாதிக் குணமதனை வகுத்துப் பாரிலுறூரைப்பேன்"

CURABLE-11

1.Thethru kuttam

2. Sadhaaru kuttam

3. Pundareega kuttam

4.Virpodaga kuttam

5. Sarmathala kuttam

6.Baama kuttam

7. Kaha nandhi

8. Venkuttam

9. Sithuma kuttam

10. Alasa kuttam

11. Vibaathiga kuttam

INCURABLE -7

1.Kabaala kuttam

2. Sarumamega kuttam

3. Kideeba kuttam

4. Avudhumbara kuttam

5. Visarchika kuttam

6. Aguvai kuttam

7. Mandala kuttam[27]

IN YUGI CHINTHAMANI-800

குட்டந்தான் பதினெட்டில் சாத்தியந்தான்

கூறக்கேள் விற்போடக பாமா குட்டம்

திட்டடந்தான் கெசசர்ம குட்டமொடு

கிருட்டிண குட்டமவுதும்பர குட்டந்தானும்

திட்டமாந் தேதிதிருக் குட்டமொடு

செய்சித்துமா குட்டங் கிடிப குட்டம்

தட்டந்தான் மிகுந்த சதாரு குட்டம்

சமகிருட்ண குட்டம் சாத்தியமா மென்னே"

CURABLE -10

1.Virpodaga kuttam

2. Baama kuttam

3. Gaja saruma kuttam

4. Krishna kuttam

5. Avuthumbara kuttam

6. Thethuru kuttam

7. Sithuma kuttam

8. Kideepa kuttam
9. Sathaaru kuttam
10. Sarmathala kuttam

INCURABLE -8

1. Pundareeka kuttam
2. Karna kuttam
3. Sikura kuttam
4. Mandala kuttam
5. Abarisa kuttam
6. Visarchika kuttam
7. Swetha kuttam
8. Vivadhika kuttam .[28]

MUKKUTRA VAERUPADUGAL (PATHOGENESIS):

Disease occurs due to the derangement in

- Uyir thathukkal
- Udal thathukkal
- Kalamarupadu (seasonal changes)
- Thinai (living lands) and
- Udal vanmai.
- Mukkutra iyal:

The function of the three uyir thathus:

a) Vali – Kattru + Veli
b) Azhal – Thee
c) Iyyam – Neer + Mann

The alteration of three thathu in their reaction to extrinsic or intrinsic factors results in disharmony. This altered harmony and balance variation of the three results in disease. Their natural ratio (1:1/2:1/4) to each other is discerned by the physician at the wrist and each nadi is individually assessed for its strength, speed and regularity.

VATHAM

The term vatham denotes vayu, dryness, pain and flatulence. Based on functions and locations it is classified into ten types. They are tabulated below.

S.No	Vatham	General Features	Changes in venpulli
1	Piranan (Uyirkkaal)	Responsible for respiration and it is necessary for proper digestion.	Normal
2	Abanan (Keel nokkukkaal)	Responsible for all the downward forces such as voiding of urine, stools, semen, menstrual flow.	Normal
3	Viyanan (Paravukaal)	Dwells in the skin and is concerned with the sense of touch, extension and flexion of the parts of the body and distribution of the nutrients to various parts of the body.	Affected (skin colour changed into white).
4	Uthanan (Melnokkukaal)	Responsible for all kinds of upward motion such as nausea, vomiting etc.,	Normal
5	Samanan (Nadukkaal)	Considered essential for proper digestion, assimilation and carries the digested nutrients to each and every organ.	Normal
6	Nagan	Helps in opening and closing of eyelids.	Normal
7	Koorman	Responsible for vision, lacrimation and yawning.	Normal
8	Kirugaran	Induces appetite, salivation, all secretions in the body including nasal secretion and sneezing.	Normal
9	Thevathaththan	Induces and stimulates a person to become alert, get anger, to quarrel, to sleep etc.,	Normal
10	Dhananjeyan	Resides in the cranium and produces bloating of the body after death. This leaves from the body after 3 days of death, forming a way through the skull[29].	Normal

PITHAM

It is the thermal life force of the body. It is sub divided into five types. They are

S.No	Pitham	General Features	Changes in venpulli
1	Anarpitham	Peps up the appetite and aids in digestion.	Normal
2	Ranjagapitham	Responsible for the colour and contents of blood.	Affected
3	Saathagapitham	Controls the whole body and is held responsible for fulfilling a purpose.	Normal
4	Pirasagapitham	Dwells in the skin and concerned with the shine, glow, texture and its complexion.	Affected (skin color Changed into white)
5	Alosagapitham	Responsible for the perception of vision[30].	Normal

KABHAM

It is responsible for the stream line functions of the body and maintains body's defence mechanism intact. It is again classified into 5 types.

S.No	Kabham	General Features	Changes in venpulli
1	Avalambagam	Lies in the respiratory organs, exercises authority over other kabhas and control the heart and circulatory system.	Normal
2	Kilethagam	Found in stomach as it seat, moistens the food, softens and helps to be digested.	Normal
3	Pothagam	Responsible for the perception of taste	Normal
4	Tharpagam	Presents in the head and is responsible for the coolness of the eyes, sometimes may be referred to as cerebrospinal fluid.	Normal
5	Santhigam	Necessary for the lubrication and the free movements of joints[31].	Normal

PARUVAKALAM

S.No	Perum pozhuthugal	Mukkutra marupaadugal
1	Kaar kaalam (Aavani & Purattasi) Mid August to Mid October	VATHAM - Vaetrunilai valarchi PITHAM – Thannilai valarchi
2	Koothir kaalam (Iypasi & Karthigai) Mid October to Mid December	VATHAM – Thannilai adaidhal PITHAM - Vaetrunilai valarchi
3	Munpani kaalam (Margazhi & Thai) Mid December to Mid February	PITHAM – Thannilai adaidhal
4	Pinpani kaalam (Masi & Panguni) Mid February to Mid June	KABHAM – Thannilai valarchi
5	Elavenir kaalam (Chithirai & Vaikaasi) Mid April to Mid June	KABHAM – Vaetrunilai valarchi
6	Mudhuvenir kaalam (Aani & Aadi) Mid June to Mid August	VATHAM – Thannilai valarchi KABHAM – Thannilai adaidhal[32]

THINAI (LAND)

Siddhars classified the lands into five types. They are

1. Kurunji – Mountain range
2. Mullai – Pastoral area of the forest
3. Marudham – The fertile river bed
4. Neidhal – The coastal region
5. Paalai – Arid desert

- Kabha diseases will occur in Kurinji land. Pitha diseases occur in Mullai land. Vadha diseases occur in Neidhal land. Staying in Paalai land is not good to health. Marudham land is the fertile area where no disease occurs. So, Marudham land is the best one to stay in.
- The winter season gives good health to the man, early summer and later rainy gives moderate health. Whereas early rainy and later summer are more prone to diseases, that's why siddhars called it as Aanaga kaalam.[33]

RELATION BETWEEN MUKKUTRAM, KAALANGAL AND THINAIGAL:

Mukkutram	**Paruvakaalam (Seasons)**			**Thinai**
	Thannilai valarchi (Accumulation)	**Vaetrunilai valarchi (Aggravation)**	**Thannilai adaidhal (Alleviation)**	
VATHAM	Mudhuvenil kaalam	Kaar kaalam	Koothir kaalam	Vatha disease is more prevalent in Neidhal land
PITHAM	Kaar kaalam	Koothir kaalam	Munpani kaalam	Pitha disease is more prevalent in Mullai land
KABHAM	Pinpani kaalam	Elavenil kaalam	Mudhuvenil kaalam[34]	Kabha disease is more prevalent in Kurunji land[35]

UDAL VANMAI (IMMUNITY)

Siddhars classify udal vanmai into three types. They are

1. Iyarkai vanmai
2. Kala vanmai
3. Seyarkai vanmai

UDAL KATTUGAL

S.No	Udal kattugal	General Features	Changes in venpulli
1	Saaram (Digestive essence)	Responsible for the growth and development. It keeps the individual in good temperament and it enriches the body.	Normal
2	Senneer (Blood)	Responsible for the color of the blood and for the intellect, nourishment, strength of the body.	Affected
3	Oon (Muscle)	Gives lookable contour to the body as needed for the physical activity. It feed the fat next day and gives a sort of plumpness to the body.	Normal
4	Kozhuppu (Fat)	Lubricates the organs to facilitate frictionless functions.	Normal
5	Enbu (Bones)	Supports and protects the vital organs, gives the definite structure of the body and responsible for the posture and movements of the body.	Normal
6	Moolai (Bone marrow)	Nourishes the bone marrow and brain which is the centre that controls other system of body.	Normal
7	Sukkilam/Suronitham (Sperm/Ova)	Responsible for reproduction[36].	Normal

PINIYARI MURAIMAI (DIAGNOSIS)

Four steps are followed in diagnosing the disease. They are

1. Poriyaal aridhal
2. Pulanal therdhal
3. Vinaadhal
4. Envagai thervugal

PORIYAAL ARIDHAL:

In this, the physician should carefully observe the changes that occur in the five sensory organs (porigal) of the patient.

PULANAL THERDHAL:

The physician carefully applies his five senses of perception, smell, taste, vision, touch and sound to understand the condition of the patient.

VINAADHAL:

The physician should interrogate about the patients name, age, occupation, socio-economic status, food habits, history of past illness, history of present illness, family history, marital status, menstrual history and frequency of pain[37].

ENVAGAI THERVUGAL:

"நாடிப்பரிசம் நாநிறம் மொழிவிழி
மலம் மூத்திரமிவை மருத்துவராயுதம்"

Nowadays advanced diagnostic tools have been developed by modern bio medical scientists. But siddhars have given eight diagnostic methodological tools. They are called as Envagai thervu.

Eight fold system of clinical assessments:

Siddhars have given eight diagnostic methodological tools. They are

1. Naadi
2. Sparisam
3. Naa
4. Niram
5. Mozhi
6. Vizhi

7. Malam
8. Moothiram[38]

GENERAL FINDINGS:

NAADI:

Naadi is responsible for the existence of life, can be felt one inch below the wrist on the radial side by means of palpation with tips of index, middle and ring finger, corresponding to vatham, pitham, kabham.

Three humours Vatham, Pitham, and Kabham are in the ratio 1:1/2:1/4 normally. Derangement in these ratio leads to various disease conditions.

Naadi nadai in venpulli

Vathapitham or Pithakabam[39]

SPARISAM:

By sparisam, the temperature of skin (thatpam- cold or veppam – heat), smoothness, roughness, sweating, dryness, hard patches, swelling, abnormal growth of organs and tenderness can be felt.

In venpulli – affected area may be or may not loss of sensation

NAA:

Signs and symptoms in the tongue are noted here. Colour, salivary secretion, ulcers, coating, inflammation, taste changes, deviation and its nature are generally noted.

- In venpulli – in anaemic conditioned tongue may be Pallor.

NIRAM:

The colour of the skin is noted here.

- In venpulli – The natural colour becomes pale or diminished

MOZHI:

Character of the speech is noted, mainly uraththa oli (high pitched), thazhndha oli (low pitched), or resembles the sound of any instrument.

- In venpulli – No changes in voice.

VIZHI:

Character of the eye is noted. Colour, warm, burning sensation, irritation, visual perception are generally noted.

- In venpulli-Not affects.

MALAM:

The stools are examined for quantity, hardening (malakattu), loose motion (bedhi), colour and smell.

- In venpulli –normal

MOOTHIRAM

a) NEERKURI (Urine examination)

Urine examination is good diagnostic method compare to naadi and other Envagai thervugal. Theraiyar mention it as.

"அருந்து மாறி ரதமும் அவிரோதமதாய்
அக்கல் அலர்தல் அகாலவூன் தவிர்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிகலசத் தாவியே காதுபெய்
தொருமுகூர்த்தக் கலைக்குட்படு நீரின்
நிறகுறி நெய்குறி நிருமித்தல் கடனே."

The early morning urine sample is collected and sample should be examined within one and hours.

SIRUNEERIN POTHU GUNAM:

"வந்த நீர்கரி எடை மணம் நுரை எஞ்சலென
றைந்தியலுளவை யறைகுது முறையே"[40]

The urine is examined for its Niram (colour), Eadai (Specific gravity), Nurai (Froth), Natram (Smell), Enjal (Deposits).

NIRAM (COLOUR)

NIRA THOGAI

" பீதம் செம்மைபைங் கருமை வெண்மையென்
றோதையங் கொழுமையை யொத்துகு நீரே.[41]

1. Yellow
2. Red
3. Green
4. Black
5. White

Urine may be any colour as mentioned above.[41]

EADAI (SPECIFIC GRAVITY)

Urine, not thick is considerably healthy. This is mentioned as

"மிகத் தடிப்பும் மிகத் தேறலும் இன்றேனில்
சுகத்தைத் தரும் மெய்ச் சுபாவ நீர் நன்றே."[42]

NURAI (FROTH)

Urine may be frothy in nature, if it is reduced in vali, azhal and ayyam are said to be deranged. This is mentioned as

“ பந்தமெய்ப் பசையிளகபடும் பருவத்
தந்தர்ப் பூதமாய் அனில மூதிரத்தில்
சம்பந்தபடும் ததினுரைப் புனலே.”[43]

NEIKURI

The early morning urine of the patient is analyzed by dropping a drop of gingely oil on the surface of the urine sample. The accumulation, formations, changes, and dispersal under the sunlight without any external disturbances of the urine sample can be noted.

The urine kept on the kidney tray in sun light, on non wind condition, should be examined by dropping a drop of gingili oil gently with rod. If oil spread like snake, it indicates vali neer; a ring indicates azhal neer and float like a pearl indicates iyya neer and sinks in urine indicates mukkutram.

“அரவென நீண்டினஃதே வாதம்
ஆழி போற் பரவின் அஃதே பித்தம்
முத்தொத்து நிற்கின் மொழிவதென் கபமே.”[44]

- Vatha neer – The oil spreads like snake
- Pitha neer – The oil spreads like ring
- Kabha neer – The oil spreads like pearl
- If the oil spreads gradually, it indicates good prognosis
- If the oil spreads fast or gets mixed completely with urine or sinks in urine, it suggests bad prognosis[43]

MANAGEMENT OF VENPULLI

REJUVENATION:

KALPA MARUNTHU

Pothu kalpam, Ponnangaani karpam (alternanthera sessilis)(Used as recipes along with milagu (piper nigvum),kariuppu(sodium chloride)).

SIRAPPU:

KITTIKIZHANGU (acalypha fruticosa) used as daily dishes like curry, vatral,etc..,

Lemon used as pickle or juices (TO BE CONTINUED FOR SIX MONTHS)

- Ayapirungaraja karpam
- Ayajambeera karpam[45]

KALPA YOGAM

- Sarvangaasana is most useful for this complained shirasana is also useful, whereas the other asanam have been included for general health and fitness.
- Pranayamam

THE COMMON BENEFITS OF YOGAM

SARVANGASANAM

USES:

It prevents narai ,thirai,and moopu .(i.e) prevents ageing .By stimulating the thyroid gland it givesa strength to all the organs of the body .it cures kutta noi

UNAVU

TO BE ADD

Bitter tasted foods

Greens

Sirukeerai(amarantus tricolor)

“kanpuhaichal……pongumpitham ……. Sirukeerai thanai kol.”

Pannai keerai (celosia argentea)-pannai yilankeerai yathu ….. karappan sirangu pun maatrum…..

Paruppu keerai (Postulaca oleraceaa)-“pillai parupilaiku pithamarmum…….”

Keerai thandu (amaranthus gangeticus)-senkeerai thandathu than theeratha pithathai thengaamal oati vidum….

TO BE AVOID

Sour ,spicy,salty foods

Curds ,oils,alcohol,sugar,non-vegetarian diet

Ulundhu(vigna mungo)

Mustard

Brinjal

OTHER ADVICES:

OLEATION:Oil bath should be taken twice a week is advisable

- Be add bitter tasted herbs like azadirachta indica, acacia catechu etc..,
- Palasu uppu (salt of butea monospermea)-For external application
- De oleaginous substance the powdered form of illupai pinnakku bassia longifolia
- Eechaam paai a type of mattress prepared from the leaves of phoenix sylvestris
- Food stuff that bring the vaatha ,piththa and kabha dhoshas to the normal physiology level have to be consumed[46] .

MODERN ASPECT

MODERN ASPECT

THE SKIN

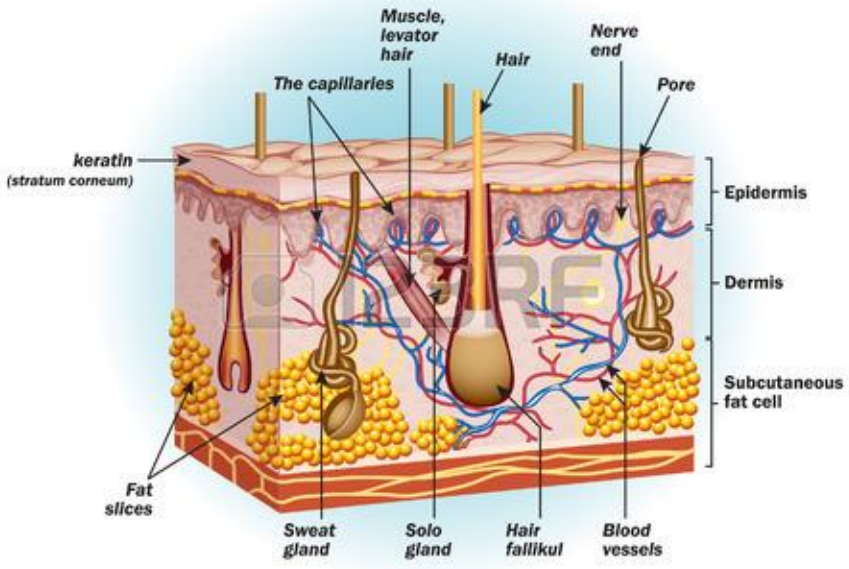
Dermatology is the study of skin disease .Disease of the skin, are acommon occurrence, account for a great deal of misery, suffering in capacity and economic loss .A very few skin disease are contagious.

Hippocrates, Father of medicine described many skin disease and divided into two groups according to their exogenous or endogenous causes. He attributed the origin of disease to abnormal mixing of black and yellow bile, blood and phlegm. The theory of abnormally mixed humors played a major role in dermatology for long time.

Dermatology is the branch of medicine dealing with skin .its roots reach back to antiquity. The obviously manifested skin disease have drawn the attention of men since time immemorial.[47]

SKIN ANATOMY

The human skin is the outer covering of the body and is continous with the mucous membranes in the region of the mouth, nose,urogenital organs and the anus .In the adult skin surfaces measures 1.5-2m while the thickness of the skin varies from fraction of a millimeter to4mm. The thickness of the epidermis varies from 0.06 -0.9 mm. The thickness of the subcutaneous fat varies considerably. Some areas are devoid of fat while in others (on the abdomen and gluteal regions), it is several centimeters thick .The mass of the skin of an adult accounts for approximately 5% While together with subcutaneous fat for about 10 to 17.7% of the total body mass.



The colour of the skin may change because the amount of the pigment in it varies due to external and internal factors. The skin surface is covered with hairs over a great area. The areas devoid of hairs are the lips, palms and soles, glans penis, inner surface of the prepuce and the inner surface of the labia majorum and minorum

Vascular System of Skin:

Vascular system of the skin is formed of several networks of blood vessels. Large arterial vessels stretch from the fascia through the subcutaneous fat and give off small branches to the fat lobules. On the boundary of the dermis and hypoderm, they divide into branches which stretch horizontally and anastomose with one another. A deep arterial plexus of skin forms, which gives rise to branches supplying the holes of the sweat glands, the hair follicles and the fat lobules. The epidermis is devoid of blood vessels.

The most powerful network of blood vessels is located in the skin of the face, palms, soles, lips, genitals and in the skin around the anus.

Lymphatic System of Skin:

The lymphatic system of the skin forms superficial and deep networks. The superficial lymphatic network arises on the papillary layer as blind rounded dilated capillaries between which there are numerous anastamoses. The second network of lymph vessels is in the lower part of the dermis and already has valves. There is a network of wide loops forming lymphatic plexus and deeper parts are continuous with lymph trunks

SKIN HISTOLOGY

The skin develops from two germinative zones .The ectoderm which is represented by the epidermis (the most superficial skin layer). And the mesoderm (the middle embroyal layer) represented by two layers namely the true skin, or dermis (the middle layer) and the subcutaneous fat or hypoderm the deepest skin layer.

The boundary between th e epidermis and dermis forms a wavy line because of the presence of skin papilla (special outgrowth on the surface of the true skin) the spaces between which are filled with epithelial processes.

LAYERS OF THE SKIN:

Skin is composed of three layers. The epidermis, dermis and the subcutaneous.

EPIDERMIS

The epidermis is defined as a stratified squamous epithelium which is about 0.1mm thick, although the thickness is greater (0.8-0.4 mm) on the palm and sole. Its prime function is to act as a protective barrier. The main cell of the epidermis is the keratinocyte which produces the protein keratin.

The four layers the epidermis represent the stage of maturation of keratin by keratinocytes.

1.Basal cell layer(Stratum basale)

2.Prickle cell layer (Stratum spinosum)

3.Granular cell layer (Stratum granulosum)

4.Horny layer (Stratum corneum)

DERMIS:

The dermis is defined as a tough supportive connective tissue matrix, containing .specialised structures .found immediately below and intimately connected with the epidermis. Two layers are distinguished in it. The papillary or sub epithelial layers and the reticular layer. The papillary layer is that part of the dermis which is found besstween the epidermis and the superficial network of blood vessel. The reticular layer merges with the subcutaneous fat is not demarcated from it sharply. The dermis is supportive connective tissue, mainly collagen, elastin and glycosaminoglycans.

SUBCUTANEOUS LAYER :

The subcutis consists of loose connective tissue and fat (upto 3 cm thick on the abdomen).

BLOOD AND LYMPHATIC VESSELS :

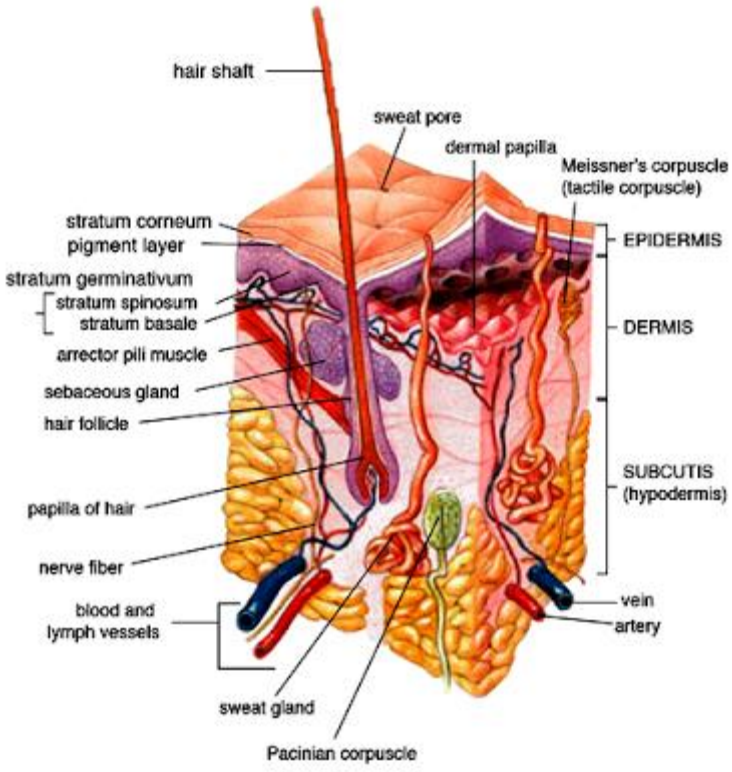
The skin also has a rich and adaptive bloodsupply. Arteries in the subcuits branchupwards,forming a superficial plexus at the papillary/ reticular dermal boundary. Branches extend to the dermal papillae .each of which has a single loop of capillary vessels , one arterial and one venous , Veins drain from the venous side of the loop to form the mid ;dermal and subcutaneous venous networks . In the reticular and papillary dermis there are arteriovenous anstomoses which are well innervated and concerned with thermoregulation.

The lymphatic drainage of the skin is important, and abundant meshes of lymphatics originate in the papillae and assemble into larger vessels which ultimately drain into the regional lymphnodes.

PIGMENTATION OF THE SKIN

The colour of the skin may be brown or even black according tothe amount of pigment present and it varies due to external and internal factors.Even in white races most parts of the skin contain brown pigment granules in the deepest layer of the germanative zone of the epidermis. In dark races they are more abundant and extend throughout the whole zone.The degree of racial pigmentation does not depend on the number of melanocytes present but on their metabolic activity and the size and shape of their melanin producing organelles the melanosomes.

Brownness of the skin depends upon the transfer of melanosomes from melanocytes into keratinocytes .Melanosomes are cytoplasmic particle formed in melanocyted and then distributed among the basal cells of the epidermis .Each melanocytes in the epidermis secrets melanosomes are site of melanin synthesis by the action of tyrosinase upon



tyrosine[48].

MELANIN

Melanin – Derived from the greek word melas, meaningblack.

Melanin is complex black – brown polymersynthesized from the amino acid dihydroxyphenyl alanine (L-DOPA).

The forms of melanin exists

- Ordinary melanin known as eumelanin and a melanin synthesized from cysteinyl DOPA with a more reddish hue, known as phaeomealnin.
- The initial part of melanin synthesis is catalysed by acopper containing enzyme complex known as tyrosinase which also catalyse the transformatiom of L.DOPA totyrosine.
- MELANIN FORMATION

Melanin, where ever it is found .is formed in the local cells by the enzyme tyrosinase (or) melanas . The mother substance, uponwhich the enzyme acts is a tyrosine derivative (DOPA) belevied to be formed in the adrenals. Melanin synthesis from the oxidation of phenylalanine or tyrosine is as follows:

1. Tyrosine → DOPA → DOPA → Quinone
2. DOPA - Quinone → 2-Carboxy2, 3 – dihydro – 5, 6 – dihyroxyindole.
→2-Carboxy – 2, 3 – dihydro – indole -5, 6 – Quinone →5, 6 Dihydroxyindole.
3. 5.6 Dihydroxyindole → Indole - 5, 6 Quinone → Melanin

Melanin formation in both human and amphibian skin is augmented bythe hormone known as intermedian or melanocyte – stimulating hormone (MSH) secreted by the pars intermedian of the pityuitary gland. Adrenocarticotrophic hormone (ACTH) secreted by anterior pituitary has melanocyte stimulating activity similar to MSH although to a much lower degree. In Addison's disease ACTH is secreted in a large amount and thereis brownish black pigmentation of the exposed parts of the skin eg, Hands,feet and mucous membrane.

Melatonin extract from bovine pineal gland, causes concentrated of melanin near the nuclei of mealnocytes in frog and asa result of this the skin becames pale. Its role in the human is not known. MSH causes the serum copper to rise and this is accompanied byin the melanin formation. Diminished formation of melanin is seen in albinism and leucoderma. In melanotic sarcoma, melanin may be found in the urine.

Distribution:

It is widely distributed in the body but peculiarly enough it is limited only to those structures which have got an ectodermal origin, for Eg: skin, hair, choroid coat of retina and substantia nigra of the brain.

Functions:

Melanin in keratinocytes is black and absorbs all visible light, The function of melanin in the choroids is namely to covert the eye ball into a perfect dark chamber. Since nervous tissue is derived from ectoderm, the melanin in the substantia nigra may represent the vestigial remnants of the melanin in the substantia nigra may represent the vestigial remnants of the melanin froming properties. Melanin is the great UVR and infrared radiation. It is also a powerful electron acceptor and may have other protective functions which as yet have been poorly characterized.

Abnormal Pigmentation:

Normal pigmentation of the skin is influenced of the amount and depth of melanin, by the degree of vascularity, by the presence of carotene and by the thickness of the horny layer. The amount of melanin produced is influenced by genetic factors, the amount and wave lengths of UV light received the amount of melanocyte. Stimulating hormone (MSH) secreted, and the effected of melanocyte stimulating chemical such as furocoumarins (psoralens) Abnormal pigmentation of the skin is produced by a variety of causes.

Types of pigmentary disorder:

Excessive pigmentation is known as hyper pigmentation and decreased pigmentation is known as hypo pigmentation. Both may be localized or generalized. In addition, increased pigmentation may result from deposits of abnormal non melanin pigments in the skin, E.g: Haemosiderin from broken down haem pigment in extravasated blood.

Homogentisic acid deposited in cartilage particular in the inherited metabolic defect known as alkaptonuria.

Drugs and heavy metals toxicity. Silver, Gold, Mercury, Arsenic poisoning, Amiodarone and phenothiazines causes slate grey, dusky skin pigmentation in exposed sites.[49]

Vitiligo:

The name 'Vitiligo' is derived from the Latin work skin eruption, victim meaning a blemish (spoil the beauty of) happens to be a synonym for it. White skin is the literal meaning of leucoderma, derma being derived from the Greek words, leucas and dermis. Leucas means white and dermis means skin.

Celeus was the first Roman physician of the 2nd century to coin the word vitiligo, because the disease resembles the white patches of a spotted calf (vitelus).

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Prevalence:

About 0.5 to 1 percent of the world's populations, or as many as 65 million people, have vitiligo. In the United States, 1 to 2 million people has the disorder. Half the people who have vitiligo develop it before age 20; most develop in before their 40th birthday. The disorder affects both sexes and all races equally; however, it is more noticeable in people with dark skin.

Epidemiology:

Vitiligo is an acquired idiopathic depigmentary condition which, though worldwide indistribution, is most common in India, Egypt and other tropical countries. It is a source of great social embarrassment of dark-skinned people. It affects all age groups with no predilection to either sex.

Gross Anatomical Changes in Vitiligo:

Vitiligo represents an acquired patchy loss of pigments of the skin. There are no gross changes seen except irregularly demarcated, Hypo pigmented patches of varying size, usually surrounded by hyper pigmented skin. These are seen distributed symmetrically or asymmetrically at various parts of the body.

I. Definition:

Vitiligo is an acquired often disfiguring, pigmentary anomaly of the skin manifested by depigmented white patches surrounded by a normal or a hyperpigmented border.As a result, white patches appear on the skin in different parts of the body. Similar patches also appear on both the mucous membranes (tissues that line the inside of the mouth and nose), and the retina (inner layer of the eyeball). The hair that grows on areas affected by vitiligo sometimes turns white.

It is an extremely common depigmentary disorder of great medicosocial significance among the dark people, etiology is uncertain association with variable penetrance; a symptomatic puncture linear, oval, circular or irregular, discrete or confluent depigmented and or hypopigmented macules on otherwise normal skin is confined to mucocutaneous functions dermatomal unilateral or bilateral, symmetrical or asymmetrical generalized or universal over laying hair retain pigment or turn white, no autonomic or sensory disturbances, sun burn or chronic solar damage in longstanding cases, impredictable and capricious course, stationary, self-healing or progressive.

It is quite clear that vitiligo is due to some derangement in the pigment metabolism resulting in appearance of white patches in the skin. It is hard to say whether the site of derangement is usually general or local, but the main affected part is the skin, which is the most exposed part of the body. It can be examined by naked eye and can furnish a lot of information about the personand the disease. In certain cases the changes are not clear. Hence the study of the skin structure and its physiology is essentials for proper assessment.

II. Etiology & Pathogenesis:

 Epidemiological studies suggest that vitiligo or a susceptibility to the disease may be inherited and about one fourth to one third of patients have family members affected with the disease. A multifactorial pattern of inheritance is revealed is most studies.

 Three possible mechanisms that may cause destruction of melanocytes, the pigment-producing cells of the skin, have been suggested by different workers.

 The**autoimmune hypothesis** originated from the observation that vitiligo is associated with some autoimmune diseases. Both cellular and humoral factors responsible for autoimmune damage to melanocytes have been demonstrated.

These autoimmune diseases include hyperthyroidism (an overactivethyroid gland), adrenocortical insufficiency (the adrenal gland does not produce enough of the hormone called corticosteroid), alopecia areata (patches of baldness), and pernicious anemia (a low level of red blood cells caused by the failure of the body to absorb vitamin B12), and it is also common in diabetes mellitus. Scientists do not know the reason for the association between vitiligo and these autoimmune diseases. However, most people with vitiligo have no other autoimmune disease.

 The **autocytotoxic or self-destruct hypothesis** suggests that some toxic molecules produced during the biosynthesis of melanin are responsible for melanocyte damage is susceptible individuals.

 The **neural hypothesis** postulates that neurochemicals liberated from nerve endings are toxic to melanocytes.

Drugs and chemicals – like quinines, guano furacin, amylphenol, chlorthiazide, broad spectrum antibiotics and chloroquin.

It is also regarded to develop through eczema scar of prick by injection needle, injury by burn or from other accidents, by friction of foot, wearing tight clothes. It has also been observed in persons who have suffered seriousillness due to typhoid, jaundice, liver diseases, diabetes, worms, constipation and diarrhea.

The non-pigmented patches whitish or reddish are round or oval in shape with smooth surface and slowly grow into large, irregularly outlined areas. It may be the result of skin diseases or it may be harmless conditions of unknown cause.

Hereditary Factors

Hereditary is one of the factors supposed to be related with venpulli Familial incidence has been reported in 7.5 to 21% in India and 33 to 40% in western countries.

Emotional Factors

It is every day knowledge and observation that emotional factors affect the skin as shown by the blushing of embracement, the pallor of fear and the pallor or redness of Change, depending on the subject and his emotional state. Experiments have demonstrated that emotional change can affect the following, which has direct relevance in the etiology of certain skin disorders.

- Control of vascularity of the skin
- Control of sebaceous gland secretion.
- Influencing the degree of oxidation.
- Influencing the tendency of pruritus.

There is due to the causative factor of this disease, venpadai from the following basic facts, it is generally considered to be a trophoneurosis. Psychological factors are known to be responsible for the precipitation and aggravation of the disease.

Others

Sometimes vitiligo can be caused by the action of monobenzyle either on hydroquinone presents in the slippers, gloves (or) other articles made of rubber or used as a depigmenting agent in the form of an irritant for pigmentary disorders. Recently vitiligo has also been observed to occur from plastic slippers as well as plastic ‘hindis’[50]

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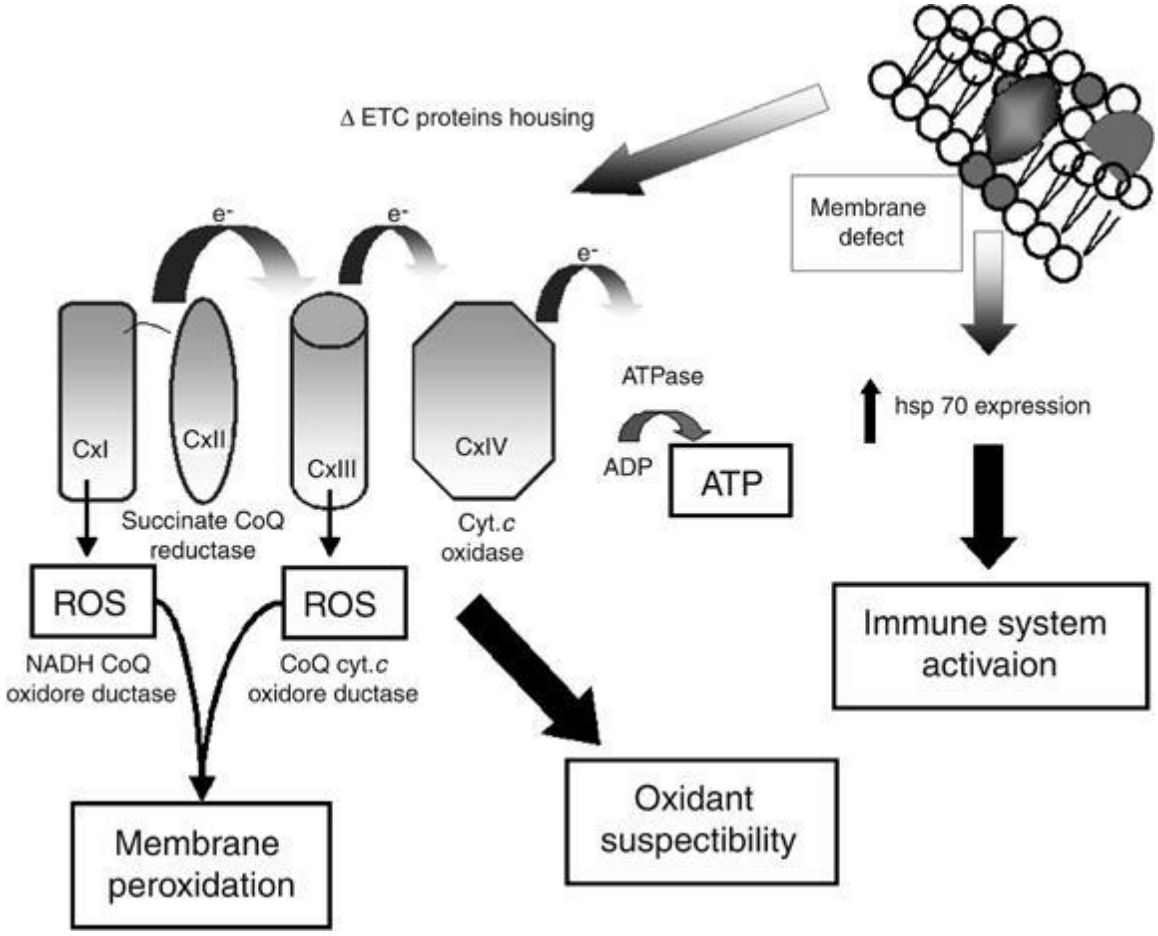
III. Pathology:

Chemically melanin pigment is a group of chromo proteins with coloured prosthetic groups, which is derived from the precursor tyrosine in the following way Tyrosine tyrinase Dihydroxy phenylanin (DOPA) Melanogenase Melanin (Dopa oxidase).

Melanin +Protein = Melano protein



The possible pathogenetic mechanism, a schematic picture of the causal and temporal sequence leading to the melanocyte functional impairment during vitiligo:

In the skin, the pigment is produced by the melanocytes of their precursor's melanoblasts. The melanoblasts are supposed to be derived from the cells of neuro ectodermal origin during the embryonic life. After birth, these cells migrate to their definitive position. The melanocytes appear as clear cells within the basal cell layer of the epidermis and show dendritic processes after special staining. These processes come in

contact with similar process of other melanocytes and epithelial cells through which the melanin pigments are donated to the basal cells of epidermis. The dermis of normal skin also shows macrophages containing melanin pigments known as melanophores, which are incapable to produce the melanin pigments.

Histopathalogic changes in Vitiligo

Marked histological changes do not occur in cases of Vitiligo. All the layers of the epidermis and dermis appear normal except a few changes which can be seen after special stains.

In the affected area the basal cells and the keratinizing cells of the other layers of epidermis do not contain melanin pigment granules in them. The contrast can be seen at a junction of the normal and vitiligenous areas of the skin, especially by silver staining or DOPA reaction. The pigment cells, the melanocytes are not seen in the affected area but they are present in the adjacent normal skin. At the border of the patches of vitiligo the melanocytes often appear large and posse's long dendritic process filled with melanin granules. Electron microscopic studies confirm the absence of melanocytes in areas of long standing vitiligo.

There are collections of mononuclear cells at dermo epidermal junctiojunction at the border between vitisliginous and normal skin. These cells are predominately small lymphocytes. In the long standing case where the skin has become thick and scaly, varying amount of the keratosis is seen.

IV. Clinical Features:

- Vitiligo affects all races with an average frequency of 1 to 2 percent of the population. Both sexes are affected equally and the disease may develop at any age. The peak age of on set in most series was between 10 and 30 years. Stressful life events or physical trauma can often precipitate the onset of disease.
- The typical macule of vitiligo is easily recognized by well-circumscribed milky white spots of varying sizes without any other discernable surface change of the skin. The hairs on the patch may turn gray or white (Leucotrichia). There may be a single spot or numerous white macules distributed all over the body. With

passage of time, the macules may enlarge and coalesce to produce extensive pigment loss. The lesions are symptomless.

 Occasionally the depigmented areas are slightly pink at the start of the disorder.

 Often the depigmented patches are symmetrical, especially when the disorder is distributed over the peripheral parts of the limbs and the face.

 These patches are more commonly found on sun-exposed areas of the body, including the hands, feet, arms, face, and lips. Other common areas for white patches to appear are the armpits and groin, and around the mouth, eyes, nostrils, navel, genitals, and rectal areas.

 In addition to white patches on the skin, people with vitiligo may have premature graying of the scalp hair, eyelashes, eyebrows, and beard. People with dark skin may notice a loss of colour inside their mouths.

 Odd patterns are sometimes noted as for example, when the depigmentation occurs over the front of the neck over the thyroid gland, or on the abdomen over the site of the pancreas or on the flanks over the sites of the adrenal glands.

 Vitiligo lesions may result from 'Koebner phenomenon' i.e., appearance of new lesions at sites of non-specific trauma such as abrasion, surgical scars, severe sunburn or inflammatory skin diseases like psoriasis or eczema.

 Vitiligo is most noticeable in the summer when the normal skin is tanned by the sun.

 Vitiligo sometimes disappears spontaneously after months or years but more usually the conditions spreads slowly and may eventually involve nearly whole of the skin.

 Early lesions may be pale white and ill defined. At this stage, Wood's lamp helps to confirm the diagnosis. Patches enlarge slowly and may affect the whole body.

V.Types:

 According to the extent of involvement and pattern of distribution, vitiligo is clinically categorized into focal, segmental, generalized, acrofacial, and universal types.

 Focal vitiligo is an isolated macule or a few macules in a localized non-dermatomal distribution.

 Segmental vitiligo is characterized by macules in a unilateral dermatomal distribution. This type of disease usually pursues a stable course.

 Generalized vitiligo is the most common type showing macules in a generalized widespread distribution. There is often striking symmetry of affection and involvement of extensor surfaces. Face (particularly around the orifices), neck, bony prominences of hands, legs; axilla and mucosal surfaces are particularly affected.

 Acrofacial vitiligo affects distal end of fingers and facial orificies in circumferential pattern.

 Universal Vitiligo implies loss of pigment over the entire body surface area with with only isolated islands of normal pigmentation remaining.

VI. Associated diseases:

 Patients with vitiligo have an increased risk of developing autoimmune diseases like thyroid diseases, Addison's disease, pernicious anemia and insulin-dependent diabetes mellitus. Auto antibodies against other organs may be detected in the absence of clinical evidence of the diseases. Premature graying of hair and alopecia areata are important cutaneous associations in some patients.

 The pigment epithelium of retina and choroid are developmentally derived from the neural crest, the cutaneous melanocytes originate from the same embryonic structure. They ma share the susceptibility to damage in vitiligo; iris and retinal pigmentary anomalies in the absence of ophthalmologic complaints may be detected in a proportion of the patients. Iris may be found in a small number of patients.

Psychosocial impact of vitiligo

 Although vitiligo by itself is symptomatic and does not cause any physical discomfort or disability, it may be associated with devastating psychological and

social consequences. Since a person's appearance is a major determinant of his/her personality traits, vitiligo, by causing cosmetic blemishes can have major impact on personality.

 Feeling of stress and embarrassment on social contacts, lack of confidence and lowered self-esteem may be detrimental to the patients, particularly when the spots are a visible area of the body.

 The psychological impact can have serious implications in deeply pigmented races such as Indians, in whom the contrast between the normally dark skin and the white lesions can be marked.

Clinical Criteria for Classification of Vitiligo:

Stages of Clinical Features

Vitiligo

Active (VI)

(i) New lesions developing

(ii) Lesions increasing in size

(iii) Border ill-defined

Quiescent (V2)

(i) No new lesions developing

(ii) Lesions stationary in size

(iii) Border hyper pigmented and well- defined.

Improving (V3)

(i) Lesions decreasing in size

(ii) No new lesions developing

(iii) Border defined and signs of spontaneous regimentation

Zosteriform:

Unilateral distribution of lesions, preferably along the course of nerves. Besides typing the stage of disease, it is useful to decide the variety (acral, Vulgaris, Zosteriform), Severity (Localized or extensive) and acuity (insidious or galloping) of vitiligo.

VII. Diagnosis:

The diagnosis of vitiligo is made based on a physical examination, medical history, and laboratory tests.

Physical examination:

White patches of skin on the body-particularly on sun-exposed areas, including the hands, feet, arms, face, and lips.

Medical History:

Important factors in the diagnosis include a family history of vitiligo; a rash, sunburn, or other skin trauma at the site of vitiligo 2 to 3 months before depigmentation started; stress or physical illness; and premature (before age 35) graying of the hair and ask whether patient or anyone his family has had any autoimmune diseases, and whether patients are very sensitive to the sun.

Laboratory tests:

To help confirm the diagnosis, we make take a small sample (biopsy) of the affected skin to examine under a microscope. In vitiligo, the skin sample will usually show a complete absence of pigment-producing melanocytes. On the other hand, the presence of inflamed cells in the sample may suggest that another condition is responsible for the loss of pigmentation.

It is usually apparent; in doubtful and early case, Wood's lamp is of great help in diagnosis.

These areas often fluorescence a golden yellow when examined under a Wood's lamp. The hypomelanotic macules in leprosy are anesthetic.

Examination of the skin in long wave UVR helps distinguish whether there is total depigmentation (as in Vitiligo) or not. It may also detect areas of depigmentation not easily seen in ordinary daylight, as well as detecting a lemon-yellow fluorescence seen in some cases of pityriasis versicolour.

VIII. Course and prognosis:

The common generalized vitiligo usually pursues a course of slow progression with enlargement of existing macules and gradual appearance of fresh spots. Quite often, after an initial phase of progression, the lesions remain relatively stable for varying periods of time only to be followed by accelerated spread, sometimes there may be very rapid spread leading to extensive loss of pigmentation within a short span of time. In an individual case the course however is unpredictable.

In comparison with the aforesaid, segmental vitiligo tends to have a very stable course. Following appearance of lesions in a dermatomal distribution, the lesions usually remain localized to the area of affection.

Spontaneous regimentation may be observed in a proportion of patients particularly in lesions on sun-exposed areas. However, the extent of spontaneous healing is seldom cosmetically significant.

It has improved considerably in recent years because of better understanding of etiological factors and advances made in therapy.

Following conditions are said to be of poor prognosis.

s1) Poor nutritional state or digestion, use of broad – spectrum antibiotics over long period. Emotional stress and nervous debility.

2) Presence of vitiligo on resistant sites like the hands and the feet, front of wrists, the elbow, the waist, the eyelids and lips.

3) Depigmented hair in vitiliginous areas.

Other Causes of Hypo Pigmentation:

Generalised depigmentation is found mostly in albinos. In this case, the characteristic dendritic melanocytes are present in the skin, but they are unable to produce melanin pigment due to defective tyrosinase activity. In albinism, the skin looks milky white, the

hairs are pale looking and the iris is transparent. The generalized pallor is also noticed in panhypopituitiarism, male eunuchoidism and phenyl ketouria.

Localised depigmentation is often noticed in the skin of pattern leucoderma. The white patches on the skin may be quite extensive and the condition is inherited as an autosomal dominant character.

Sometimes sharply defined focal depigmented areas are found on skin of persons suffering from vitiligo. In the affected areas, melanocytes are absent and there is no trace of melanin. The condition is an acquired one and shows some familial tendency. [51]

Leucoderma:

Leucoderma may be defined as a type of acquired skin depigmentation produced by some specific substances (or) dermatosis several types of Leucoderma may be seen

1. Occupational Leucoderma may occur in those who work in rubber garments (or) wear gloves that contain antioxidant monobenzyl ether of hydroguinone many phenolic compounds can produces Leucoderma.
2. Postinflammatory Leucoderma may result from many inflammatory dermatoses such are, Pityriasis rosea, psoriasis, herpes zoster, secondary syphilis, and morphea.

Burns, scars post dermabrasion and intralesional steroid injections with depigmentation are other examples of Leucoderma

Leucoderma is also commonly seen on the flanks of ladies wearing tight petticoat strings where the prolonged pressure is presumed to lead to depigmentation.[52]

Piebaldism:

In this condition there is a white forelock and white patches on the skin surface. In waardenburgs syndrome the condition associated with sensory deafness.

Pityriasis Versicolour	Superficial fungus infection leading to disturbance in pigment production, common multiple pale scaling patches on trunk
Pityrisis alba	Mild patchy eczema of the face in children causing a disturbance in pigment production.
Leprosy	One or several paler macules on trunk or limbs that are hypo aesthetic.
White macules of affecting tuberous sclerosis	Uncoming development of anomaly of CNS, connective tissue and skin; several “maple leaf” shaped hypopigmented macules.
Postinflammatory hypopigmentation	After inflammatory skin disease (after eczema or trauma to the skin; irregular in shape and in depth of pallor).
Naevous anaemicus	Rare developmental solitary white patch usually on trunk; thought to have vascular basis
Chemical toxicity	May look very much like vitiligo; seen in workers in rubber industry exposed to parateriary benzyltoluence.

DIFFERENTIAL DIAGNOSIS OF THE IMPORTANT DEPIGMENTARY DISORDERS

Distinguish features	**Albinism**	**Naevus Depigmentosus**	**Vitiligo**	**Leprosy**	**Pityriasis**
Age	Congenital present at birth	Congenital present at birth	Acquired	Any age	Any age
Distribution	Complete (or) Partial	Unilateral	Any area	Any area	Trunk, Neck, and Face
Course	Stationary	Does not increase in size or changing shape	Progressive	Progressive	Progressive; worse in mansoon and summer
Hyper pigmentary border	Nil	Nil	Present	Inflammatory	Nil
Heredo family	Hereditary	No thereditary	Rare	Nil	Nil
Other features	Hair and eyes may be affected	Nil	Hair may be affected	Anesthesia thickened nerves, nasal, bleeding slit smear and biopsy	Furfuraceous like dandruff, scaling in head macules and large patches [53]

VITILIGO AREA SEVERITY INDEX (VASI):

Its name is an adoption from PASI score in psoriasis. The percentage of vitiligo involvement is calculated in terms of hand units. One hand unit is approximately equivalent to 1% of the total body surface area. The degree of pigmentation is estimated to the nearest of one of the following percentages: 100% - complete depigmentation, no pigment is depigmentation]present; 90% - specks of pigment present; 75% - depigmented area exceeds the pigmented area; 50% - pigmented and depigmented areas are equal; 25% - pigmented area exceeds depigmented area; and 10% - only specks of depigmentation present [1,3]. The VASI for each body region is determined by the product of the area of vitiligo in hand units and the extent of depigmentation within each hand unit measured patch. Total

body VASI = S All body sites [Hand Units] ´ [Residual depigmentation] [1].

The VASI for each body region is determined by the product of the area of vitiligo in hand units and the extent of depigmentation within each hand unit measured patch.

Total body VASI = S All body sites [Hand Units] ´ [Residual

VASI score	~−50	Very much worse
VASI score	-50 -25	Much worse
VASI score	-26 -10	Worse
VASI score	-10 0	Minimally worse
VASI score	0 10	Minimally improved
VASI score	10 25	Improved
VASI score	25 50	Much improved

VASI score	+50~	Very much improved

CALCULATION:

The body is divided into four sections (head (H) (10% of a person's skin); arms (A) (20%); trunk (T) (30%); legs (L) (40%)). Each of these areas is scored by itself, and then the four scores are combined into the final PASI. For each section, the percent of area of skin involved, is estimated and then transformed into a grade from 0 to 6:

- 0% of involved area, grade: 0
- < 10% of involved area, grade: 1
- 10–29% of involved area, grade: 2
- 30–49% of involved area, grade: 3
- 50–69% of involved area, grade: 4
- 70–89% of involved area, grade: 5
- 90–100% of involved area, grade: 6

Within each area, the severity is estimated by three clinical signs: erythema (redness), induration (thickness) and desquamation (scaling). Severity parameters are measured on a scale of 0 to 4, from none to maximum.

The sum of all three severity parameters is then calculated for each section of skin, multiplied by the area score for that area and multiplied by weight of respective section (0.1 for head, 0.2 for arms, 0.3 for body and 0.4 for legs).

TRIAL DRUG

TRIAL DRUG

இரசச்சீனீச் சூரணம்

கண்டிட்ட ரசச்சீனீச் சூரணத்தைகேளு

காணுரசஞ் சுத்திசெய்துபல முக்கால்தான்

விண்டிடவே **கெந்தி**யொரு பலந்தானப்பா

மேலான **அரிதாரம்** பலந்தான்ரெண்டு

பண்டிட்ட **நற்பரங்கிபட்டை** பலம்ணாலு

பாரடா **கொடிவேலி**வேரின்பட்டை

துண்டிட்டு ஒன்றரைதான் பலமேபோடு

சொல்லாதெ எட்டிவிதைபலந்தா ஒன்றே

பலத்தாலே நிறுத்து கல்லுரலிலிட்டு

பகருஞ் **சேங்கொட்டை** ஐந்து வறுத்துப்போடு

பெலத்தாலே இடித்துச் சூரணமே செய்து

பேனியே அந்திசந்தி இளவெந்நீரில்

நலத்கதாலேஒருபொழுதுக்கரைக்கழஞ்சி

நல்கிடவே பத்தியத்தை நாட்டக்கேளு

கோழியொடு துவரையு மூர்க்குருவியாமே

ஆகாது புளீயுப்பு கசப்பு மூன்றும்

அப்பனேபதினைந்து தினம் போமட்டும்

ஏகாத பிணீயகலும் வகையைக்கேளு

ஏழுமூலங் கருங்குட்ட மண்டவாதம்

போகாத செங்குட்டம் **வெண்குட்டங்கள்**

புருடருட பிளவைதண்டுக் கிரந்திப்புண்கள்

சாகாத அரையாப்புச் சூலையெல்லாந்

தான்கெட்டே னென்றுப் போந்தான் கண்டீரே.

அகத்தியர்வல்லாதி 600 (பக்கஎண்:119-120)[54]

INGREDIENTS

RASACHEENEE CHOORANAM

***1*. Suthitha Rasam -**Hydragyrum - (Mercury Quick Silver) -3/4 Palam(28 gm)

2. Suthitha Ganthagam - (Sulphur**)-1palam (35 gm)**

3. Suthitha Thalagam - Arsenic (Arsenic-orpiment/ Yellow Arsenic Trisulphide)- **2palam(70 gm)**

4. Parangipattai - (Smilax China) -4palam (140 gm)

5. Kodiveli Vearpattai- (Plumbago Indica)-1 1/2palam(52.5gm)

6. Eattivithai - (Strychnos Nux Vomica) - 1palam (35 gm)

7. Searangkottai - (Semicarpus Anacardium) -5numbers

ACTIONS OF TRIAL DRUGS:

S.No	Drugs	Botanical name	Actions
1	Parangippattai	*Smilax china.linn.*	Alternative Antisyphilitic Aprodisaic Depurative[55]
2	Etti	*Strychnos nux –vomica .linn*	Antiseptic Tonic Diuretic Stimulant Carminative Purgative[56]
3	Kodiveli vearpattai	*Plumbago indica*	Tonic Stomachic[57]
4	Serangkottai	*Semecarpus anacardium.linn.f*	Alterative Caustic[58]
5	Rasam	*Hydrargyrum(mercury of quicksilver)*	Diuretic Sialagogue Tonic Alterative Antibilious[59]
6	Ganthagam	*sulphur*	Cholagogue Diaphoretic Alterative[60]
7	Thalagam	*Yellow arsenic trisulphide*	Expectorant Alterative Tonic Antidote[61]

சேராங்கொட்டை பொதுகுணம்:

குட்டம் கயரோகங் கொல்லும் விடபாகந்

துட்டந்தரு கிருமி சூலையும் போம் மட்டலருங்

கூந்தன்மயிலே கிரந்திக் குட்டம்போஞ் செங்கையில்

ஏந்து சேங்கொட்டை தனையே.[62]

கந்தகம் பொதுகுணம்:

நெல்லிக்காய்க் கந்திக்குநீள் **பதினெண்குட்ட மந்தம்**

வல்லை கவிசை குன்மவாயு கண்ணோய் – பொல்லா

விடக்கடிவன் மேகநோய் வீறுசுரம்பேதி

திடக்கிரசுணீ கபம் போந்தேர்.[63]

இரசம் பொதுகுணம்:

விழிநோய் கிரந்தி குன்மம் மெய்ச்சூலை புண்குட்

டழிகாலில் விந்துவினால் அத்தை – வழியாய்

புரியு விதியாது புரியினோ யெல்லாம்

இரியு விதியாதுமில்.[64]

STANDARD OPERATIVE PROCEDURE

INTERNAL MEDICINE

Purified herbo mineral ingredients (except semecarpus) are finely powdered into the stone morter then properly purified semecarpus add to the above powder, mixed together well and bottled up.

EXTERNAL MEDICINE

Karkadagasingi powder is mixed with kaadi and apply to the affected area.

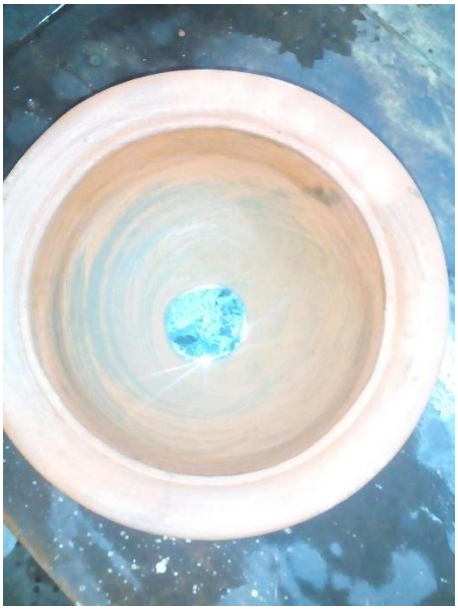
DOSAGE:10 mg twice aday

ADJUVANT: Palm jaggery

DURATION: 48 Days,

INGREDIENTS OF TRIAL DRUG

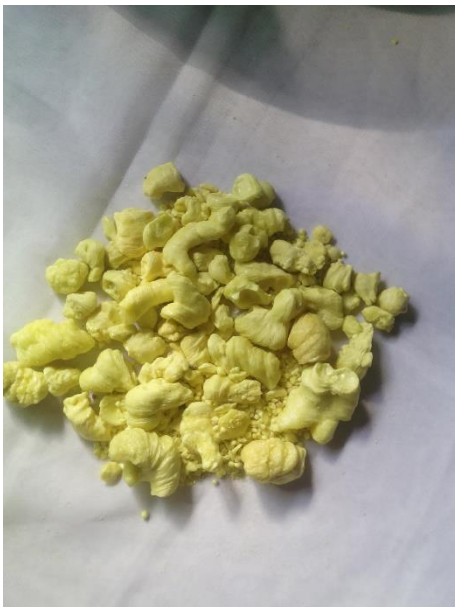
சுத்திக்கு முன்-இரசம்



சுத்திக்கு பின்-இரசம்

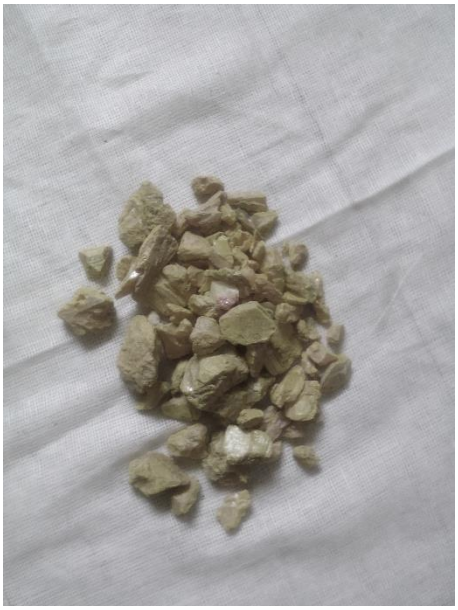
சுத்திக்கு முன்-கந்தகம்

சுத்திக்கு பின்-கந்தகம்

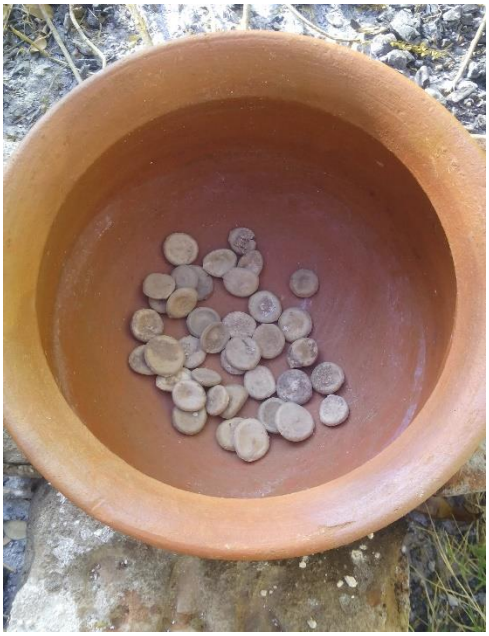


சுத்திக்கு முன்-தாளகம்

சுத்திக்கு பின்-தாளகம்



எட்டி



பறங்கிப்பட்டை

கொடிவேலி வேர்ப்பட்டை

சேராங்க்கொட்டை

கர்கடகசிங்கி

காடி

இரசச்சீனீ சூரணம்

MATERIALS AND METHODS

MATERIALS AND METHODS

PROTOCOL

Study Design

An open pilot study on Venpulli was carried out in the post graduate department of maruthuvam in Govt.Siddha Medical College attached to Arignar Anna Hospital of Indian Medicine, Chennai – 106 during the period of 2014 – 2016.

The study was approved by **Instititional Ethics Committee (IEC)** and the approval number is **GSMC-CH-ME-2/001/2014**. It was registered in **Clinical Trials Registry – India (CTRI)** and the register number is **CTRI/REF/2016/06/011520**.

Sample size

The study is conducted in 20 selected venpulli patients of both genders between age groups of 18 to 60 years.

Selection Criteria

The patients having following parameters are selected for the study.

- Age: Between 15 to 60 years
- Sex: Both male and female.
- Hypo pigmented patches with hyper pigmented border without any structural changes in any part of the body
- Willing to co operate for taking photographs whenever required with his consent

Exclusion Criteria

- Albinism
- thyroid disorder
- Leprosy
- STD
- HIV

- Burns
- Pregnancy and lactation.
- Fungal infestation
- Patient with lesion in the lips ,sole , palm and genitalia

Proforma

The case sheet proforma for venpulli was prepared based on Siddha diagnostic methodology with necessary modern techniques.

History taking

For better treatments and results a detailed clinical history was taken regarding present illness, past illness, family history, menstrual history, occupational history, socio economic status, residential area, etc.,

Investigation

All patients were screened by the following investigations. This was carried out regularly before and after treatment.

- **Blood for biochemical examination**

  The blood was tested for sugar, urea, serum creatinine to know the renal function and its excretion.
- **Urine Examination**

  Albumin, Sugar, Deposits.
- **Thyroid profile**
- **Liver function test**
- **Renal function test.**

Drug and dose schedule:

Internal Medicine-Rasacheenee Chooranam – 10mg, bd after food palm jaggery for 48 days.

External Medicine – Karkadagasingi pattru

RESULTS

AND

OBSERVATION

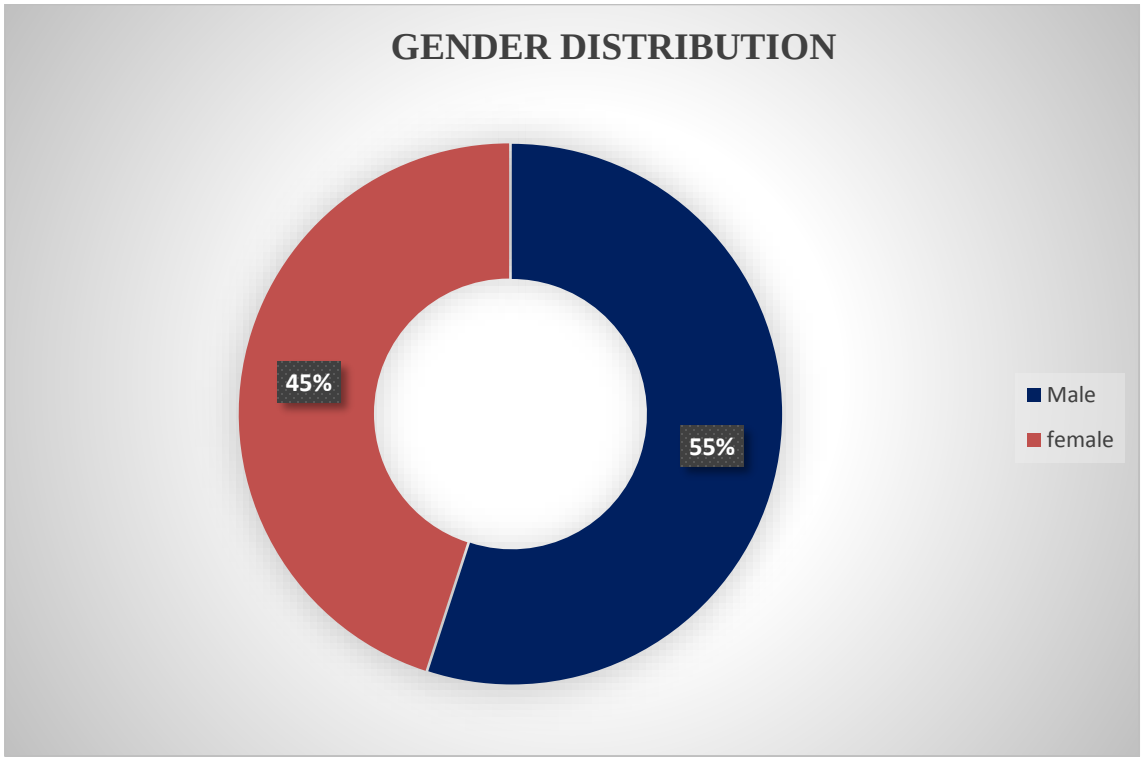
RESULTS AND OBSERVATION

20 cases having venpulli were selected and treated in OPD of PG maruthuvam department attached to AAGHIM, Chennai – 106 during the year 2014 – 2016. The result and observation during that clinical study are as follows.

- Gender distribution
- Age distribution
- Occupation
- Socio- economic status
- Etiology Reference
- Dietary habits
- Seasonal occurence
- Distribution of Thinai
- Site of lesion
- Duration of disease
- Distribution of mukkutram – vatham
- Distribution of mukkutram – pitham
- Distribution of mukkutram – kabham
- Ezhu udalthathukkal
- En vagaithervugal
- Naadi
- Neikuri
- Clinical features
- Clinical improvement in VASI score.
- Grading of Result.

GENDER DISTRIBUTION

S.No	GENDER	NUMBER OF CASES	PERCENTAGE (%)
1	Male	11	55%
2	Female	9	45%

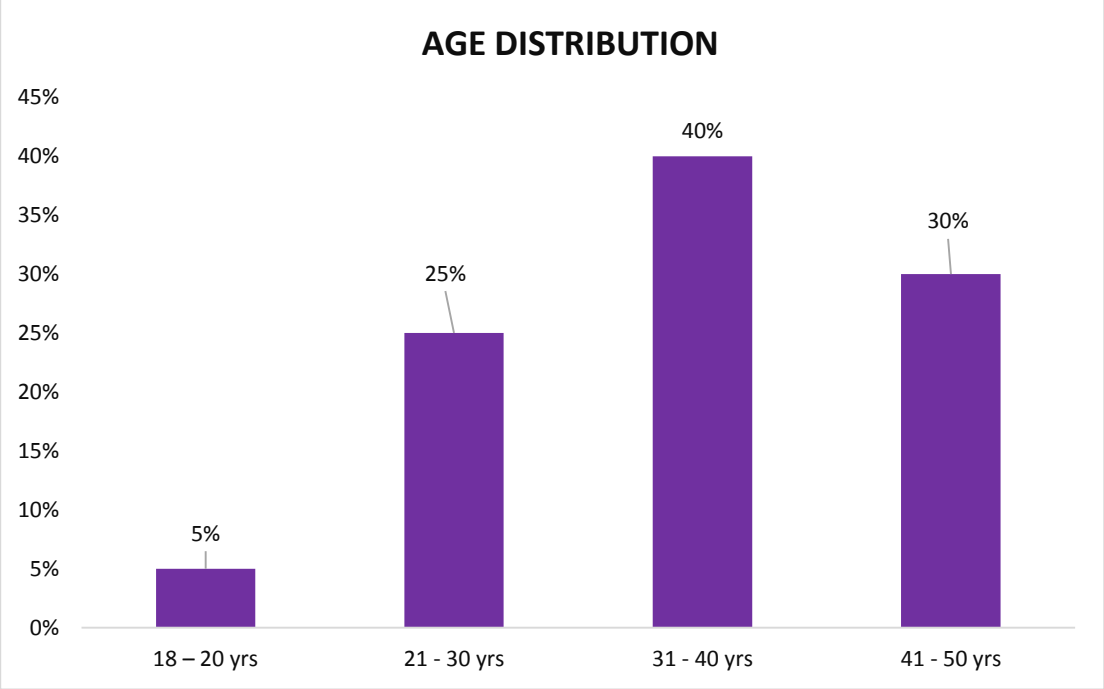


INFERENCE:

About 55% were males and 45% were females

AGE DISTRIBUTION

S.No	AGE IN YEARS	NUMBER OF CASES	PERCENTAGE (%)
1	18 – 20 yrs	1	5%
2	21 - 30 yrs	5	25%
3	31 - 40 yrs	8	40%
4	41 - 50 yrs	6	30%

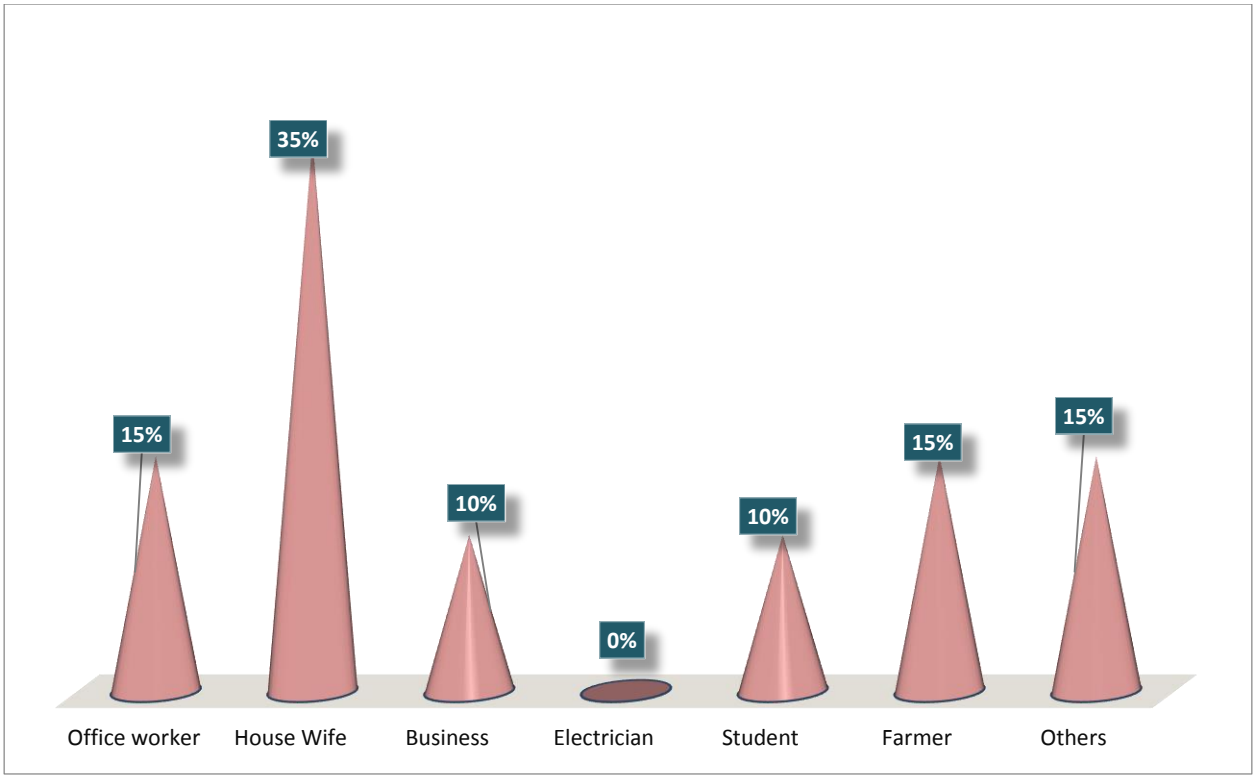


INFERENCE

Majority of the case that is 40% were in the 3rd decade, 30% were in the 4th decade, 25% were in the 2nd decade, 5% were in the 1st decade

OCCUPATION

S.No	OCCUPATION	NUMBER OF CASES	PERCENTAGE (%)
1	Office worker	3	15%
2	House Wife	7	35%
3	Business	2	10%
4	Electrician	0	0%
5	Student	2	10%
6	Farmer	3	15%
7	Others	3	15%

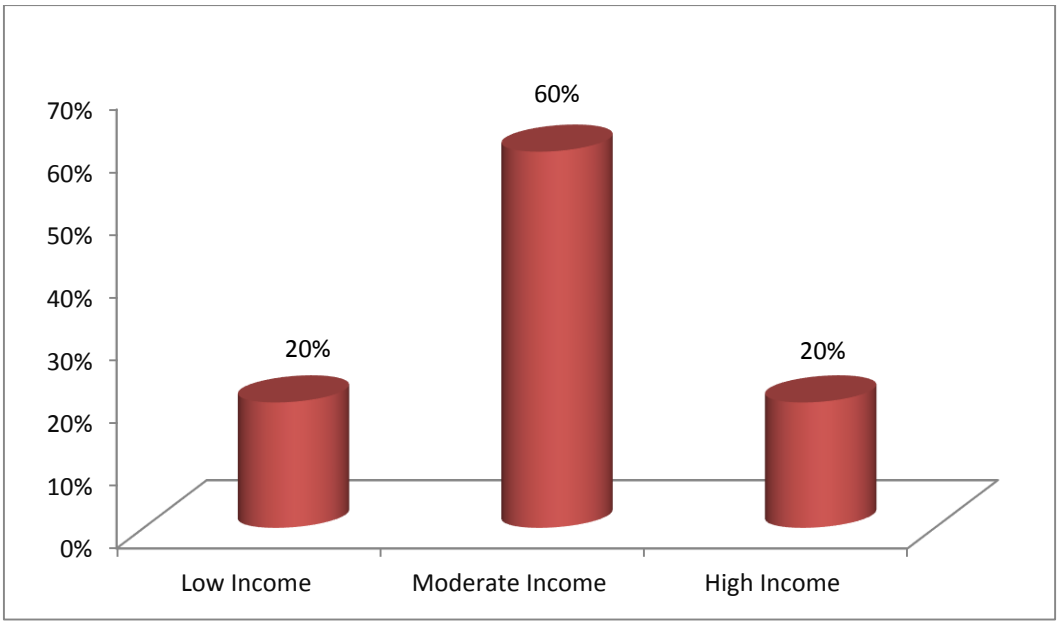


INFERENCE

0ut of 20 patients (100%), 35% were house wife,15% were office worker, 15% were farmer, 0% were electrician, 10% were business ,10% were student, 15% were in other occupation.

SOCIO – ECONOMIC STATUS

S.No	SOCIO – ECONOMIC STATUS	NUMBER OF CASES	PERCENTAGE (%)
1	Low Income (below 25,000 per annum)	4	20%
2	Moderate Income (25,000 – 50,000 per annum)	12	60%
3	High Income (Above 50,000 per annum)	4	20%

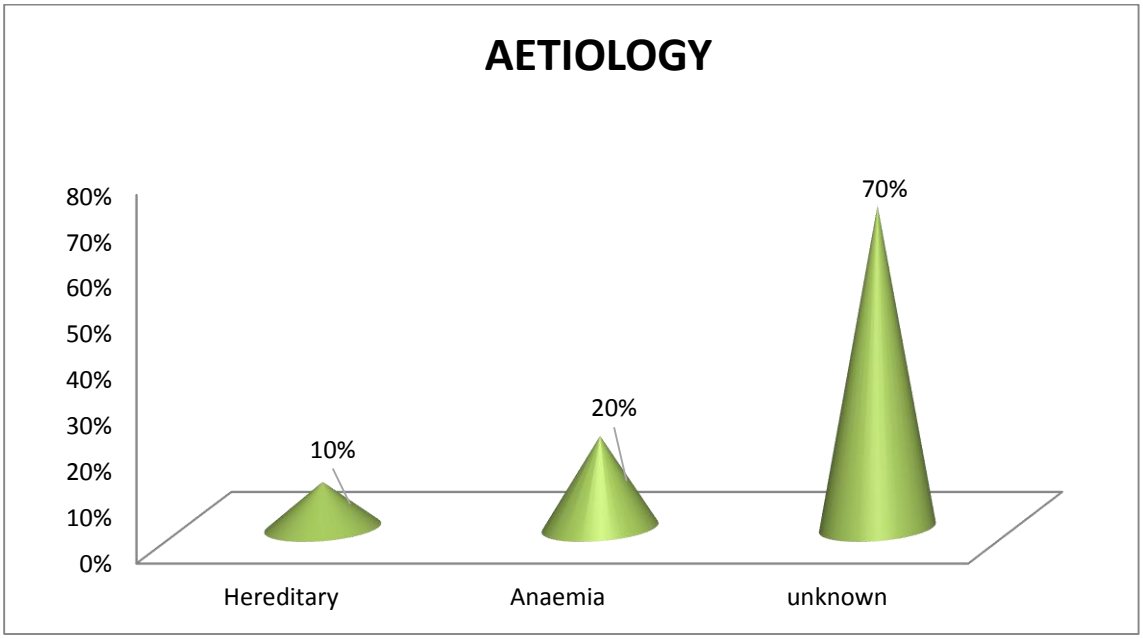


INFERENCE:

Among 20 cases 20% comes under low economic status, 60% of them under moderate status and 20% of them under high income status.

ETIOLOGY REFERENCE

ETIOLOGY	NO OF CASES OUT OF 20	PERCENTAGE
Hereditary	2	10%
Anaemia	4	20%
unknown	14	70%

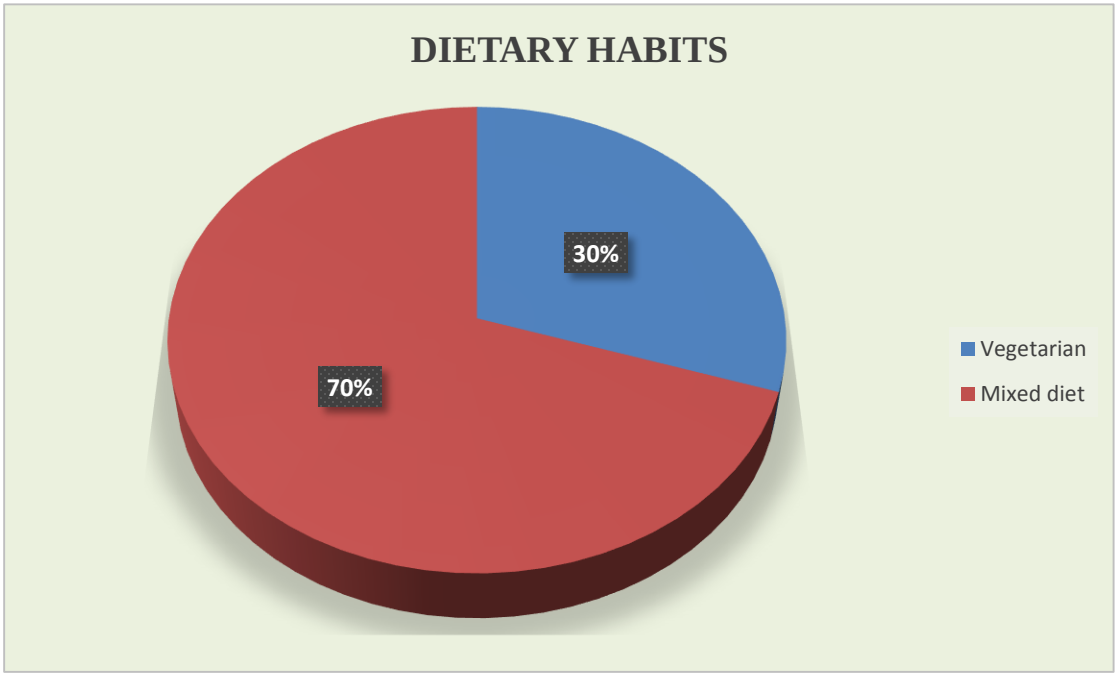


INFERENCE

Among 20 patients, 2 patients (10%) had Hereditary Etiology,4 patients (20%) were Anaemia and 14 patient had unknown causes (70%).

DIETARY HABITS

S.No	DIET	NUMBER OF CASES	PERCENTAGE (%)
1	Vegetarian	6	30%
2	Mixed diet	14	70%

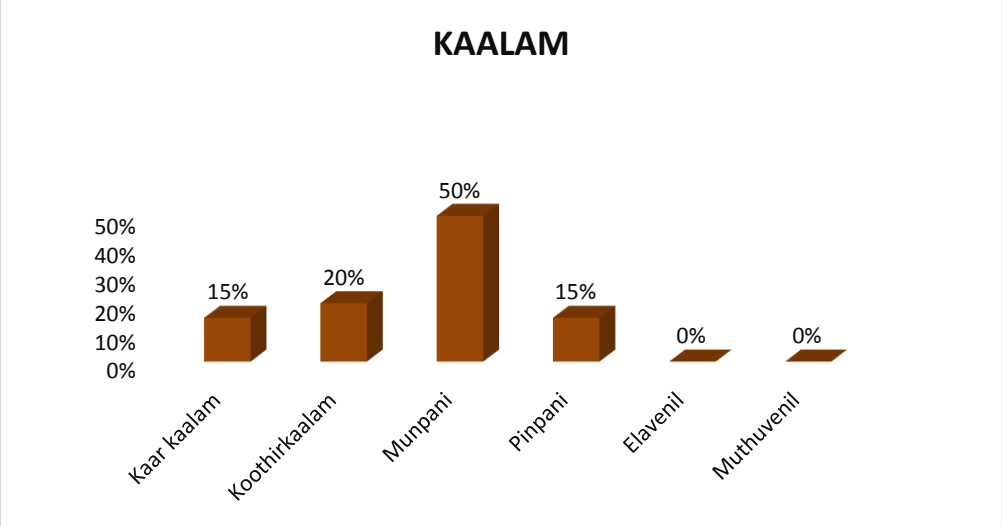


INFERENCE

Among 20 patients, 6 patients (30%) were taking vegetarian food and 14 patients (70%) were taking mixed diet.

SEASONAL OCCURENCE

S.No	KAALAM (Season)	NUMBER OF CASES	PERCENTAGE (%)
1	Kaarkaalam (Mid Aug – Mid Oct)	3	15%
2	koothirKaalam (Mid Oct – Mid Dec)	4	20%
3	Munpanikaalam (Mid Dec – Mid Feb)	10	50%
4	Pinpanikaalam (Mid Feb – Mid Apr)	3	15%
5	Elavenirkaalam (Mid Apr – Mid Jun)	0	0%
6	Muthuvenirkaalam (Mid Jun – Mid Aug)	0	0%

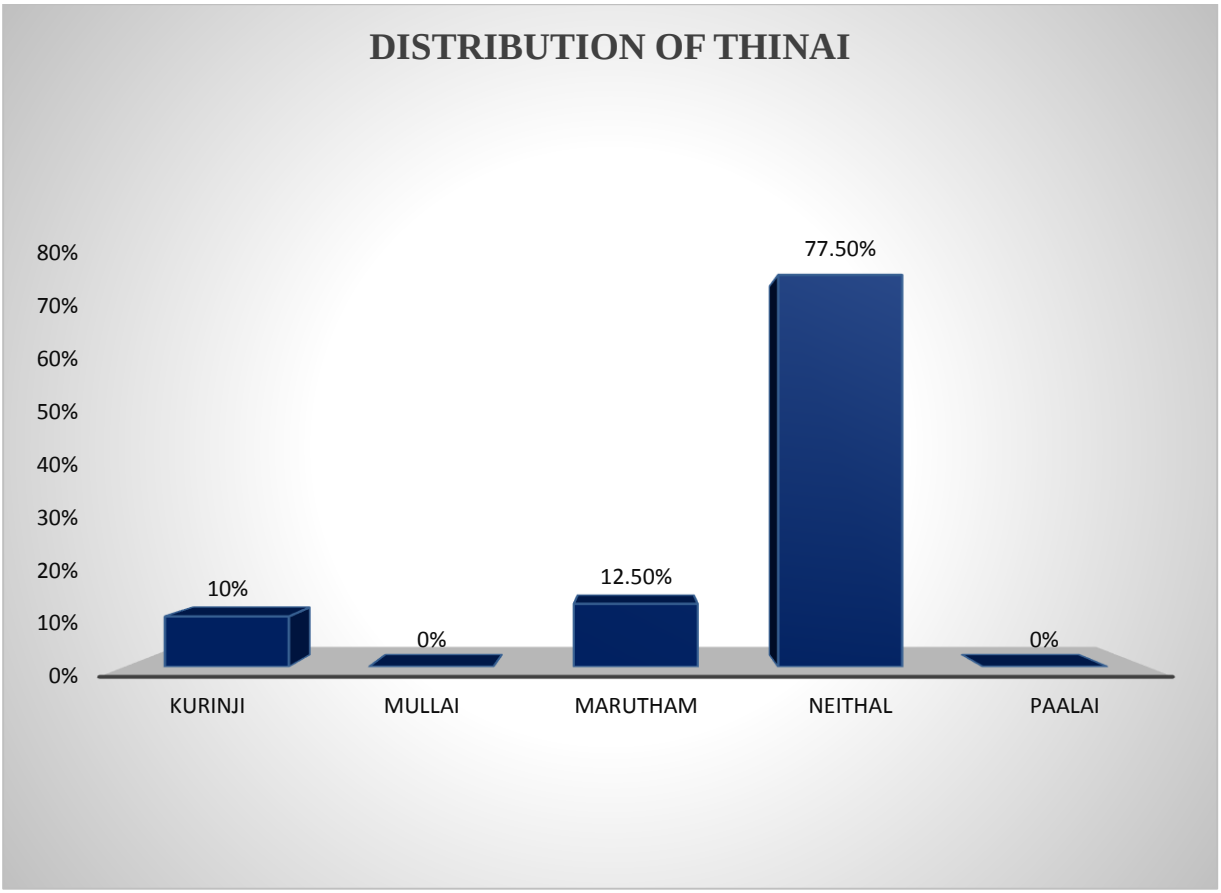


INFERENCE

According to paruvakaalam highest incident of 10 cases (50%) were noted in munpanikaalam and 4 cases (20%) were noted in Koothirkaalam, 3 cases (15%) were noted in kaarkaalam, 3 cases (15%) were noted in Pinpanikaalam.

DISTRIBUTION OF THINAI

S.No	THINAI	NUMBER OF CASES	PERCENTAGE (%)
1	Kurinji	4	10%
2	Mullai	0	0%
3	Marutham	5	12.5%
4	Neithal	11	77.5%
5	Paalai	0	0%

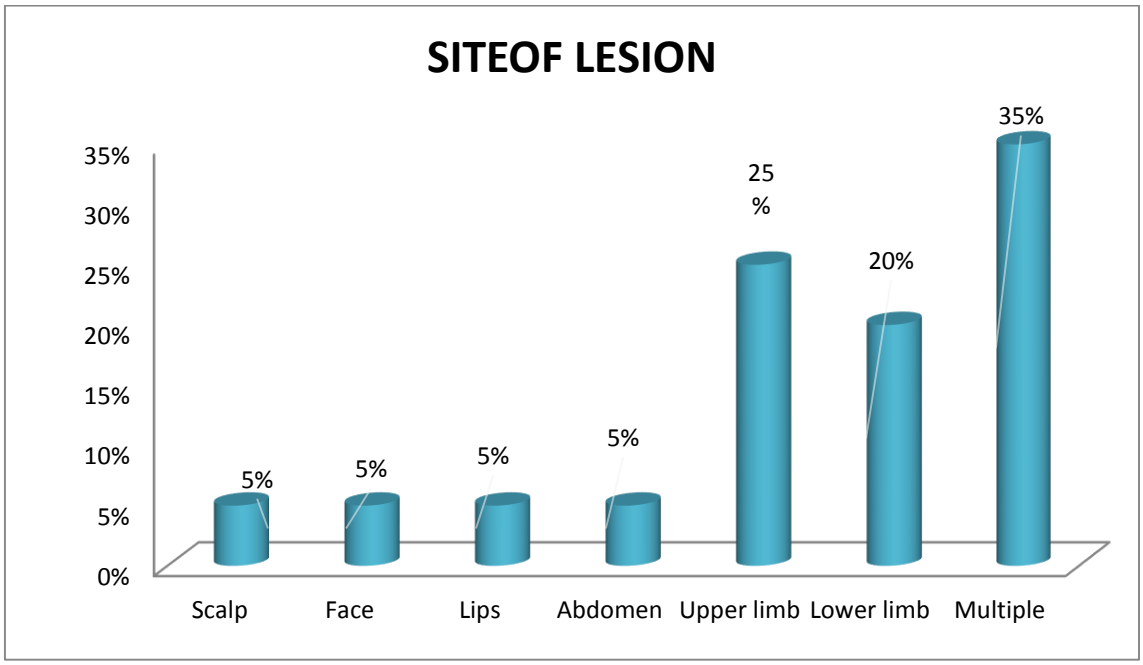


INFERENCE

According to thinai the highest distribution 77.5% was noted in neithal, 12.5% in marutham, and 10% in kurinji

SITE OF LESION

SITE OF LESIONS	NO OF CASES OUT OF 20	PERCENTAGE (%)
Scalp	1	5%
Face	1	5%
Lips	1	5%
Abdomen	1	5%
Upper limb	5	25%
Lower limb	4	20%
Multiple	7	35%

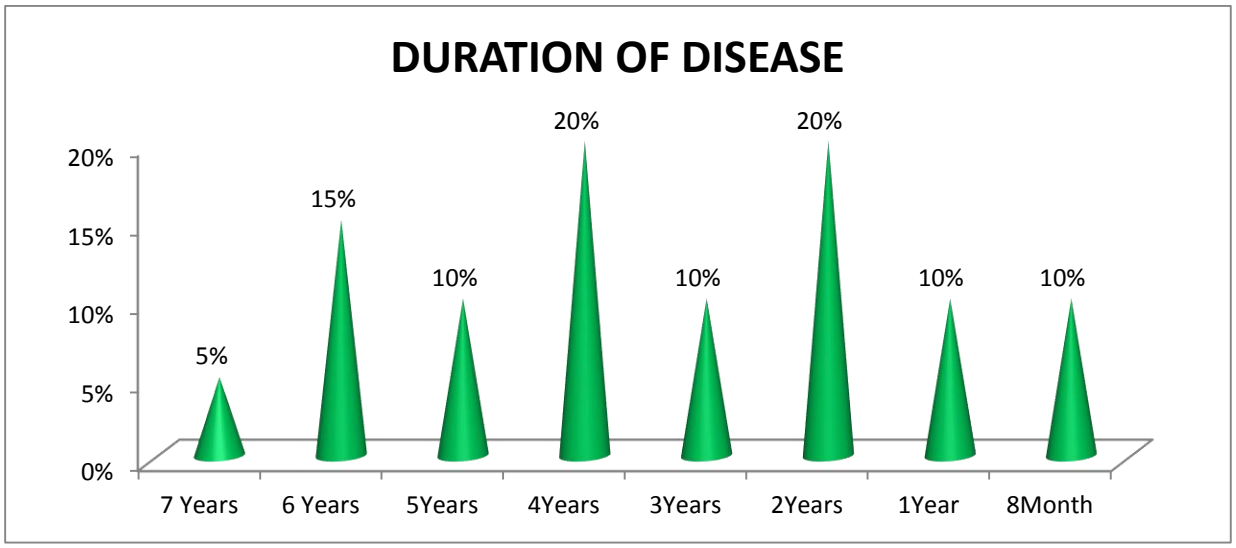


INFERENCE:

According to the site of lesion,Out of 20 patient ,1 patient(5%) had scalp region lesion,1 patient (5%) had face region lesion, 1 patient(5%) had lips region lesion,1 patient (5%) had abdomen region lesion,5 patient (25%) had upper limb region lesion ,4 patient (20%) had lower limb region lesion,7 patient (35%) had multiple region lesion.

DURATION OF DISEASE

DURATION OF DISEASE	NO OF CASES	PERCENTAGE
7 Years	1	5%
6 Years	3	15%
5Years	2	10%
4Years	4	20%
3Years	2	10%
2Years	4	20%
1Year	2	10%
8Month	2	10%

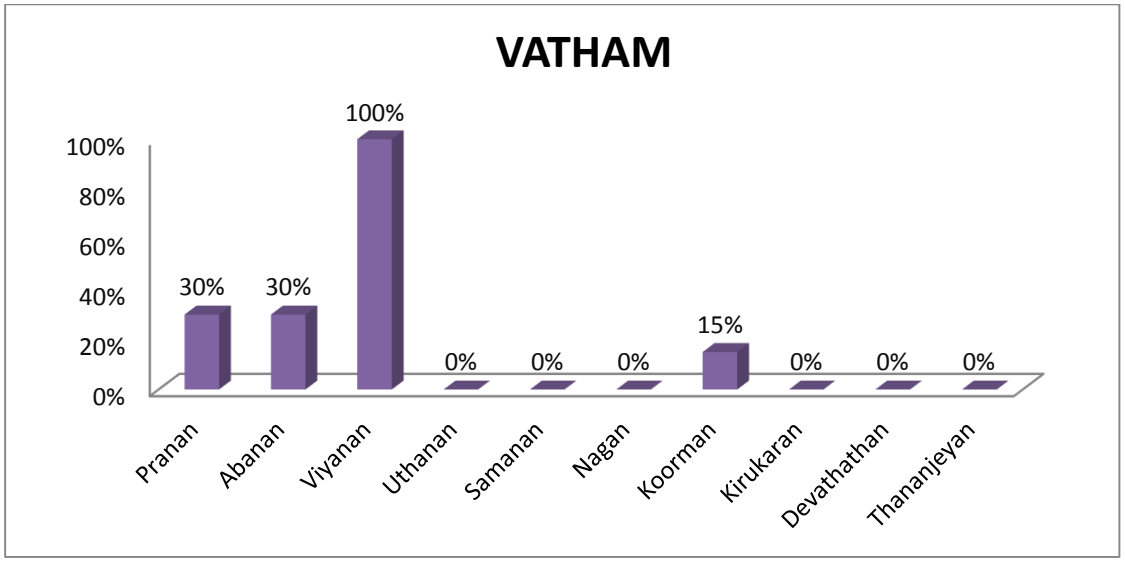


INFERENCE:

According to the duration of disease ,out of 20 patient , 1patient (5%) had 7 year of disease,3 patient (15%) had 6 year of disease ,2(10%) patient had 5 year of disease,4 patient (20%) had 4 year of disease,2 patient (10%)had 3 year of disease,4 patient (20%) had 2 year of disease,2 patient (10%) had 1 year of disease, 2 patient (10%) had 8 month of disease.

DISTRIBUTION OF MUKKUTRAM – VATHAM

S.No	VATHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Pranan	6	30%
2	Abanan	6	30%
3	Viyanan	20	100%
4	Uthanan	0	0%
5	Samanan	0	0%
6	Nagan	0	0%
7	Koorman	3	15%
8	Kirukaran	0	0%
9	Devathathan	0	0%
10	Thananjeyan	0	0%

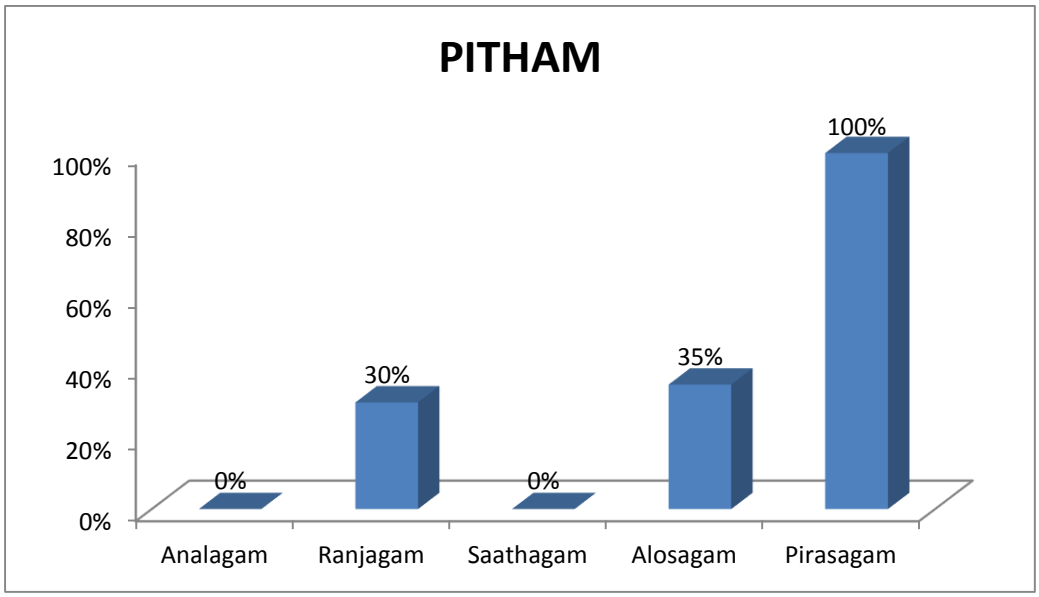


INFERENCE

Out of 20 patients pranan was affected in 6 patients (30%),Abanan was affected in 6 pateints (30%),Viyanan was affected in 20 patients (100%), and Koorman was affected in 3 patients (15%).

DISTRIBUTION OF MUKKUTRAM – PITHAM

S.No	PITHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Analagam	0	0%
2	Ranjagam	6	30%
3	Saathagam	0	0%
4	Alosagam	7	35%
5	Pirasagam	20	100%

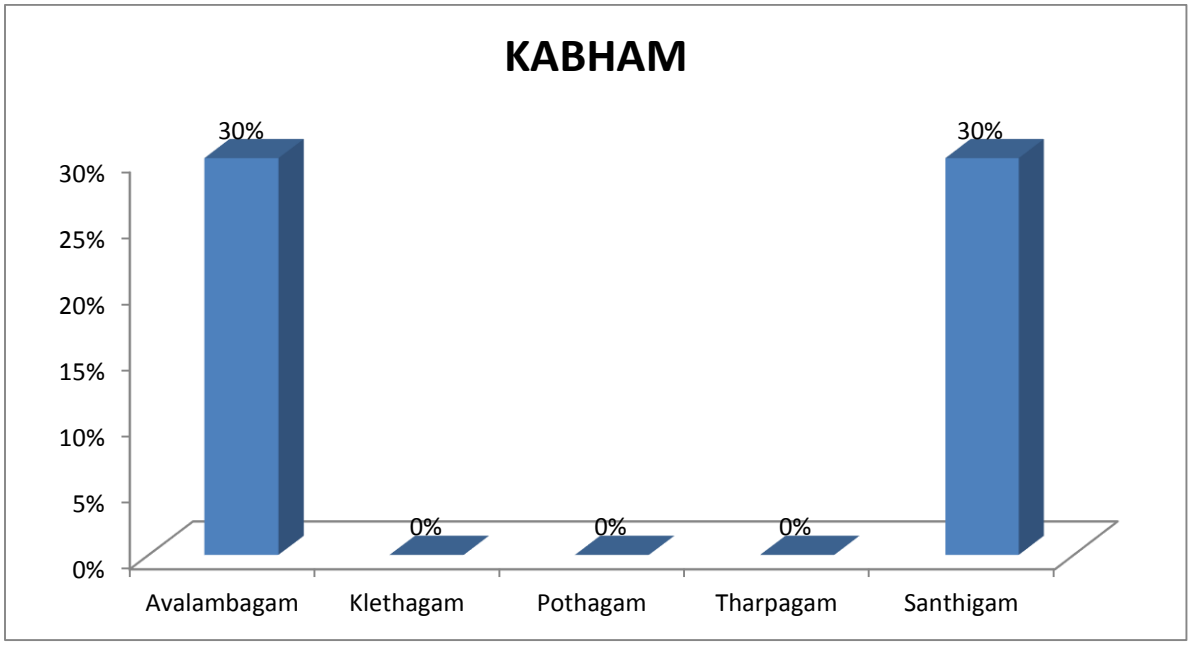


INFERENCE

Out of 20 patientsAnalagam was affected in 0 patients (0%), Ranjagam was affected in 6patients (30%), Sathagam was affected in 0 patients (0%), Aalosagam was affected in 7patients (35%),pirasagam was affected in 20 patients(100%)

DISTRIBUTION OF MUKKUTRAM – KABHAM

S.No	KABHAM	NUMBER OF CASES	PERCENTAGE (%)
1	Avalambagam	6	30%
2	Klethagam	0	0%
3	Pothagam	0	0%
4	Tharpagam	0	0%
5	Santhigam	6	30%

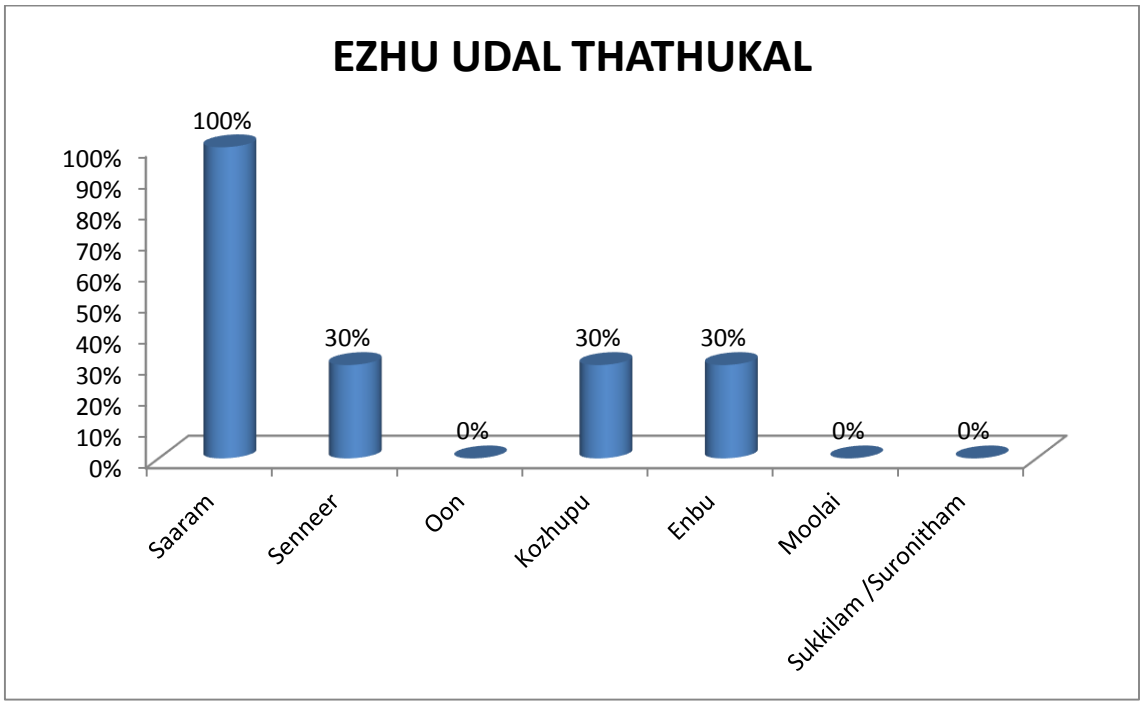


INFERENCE

Out of 20 patients, Avalambagam was affected in 6 patients (30%), Santhigam was affected in 6 patients (30%).

EZHU UDAL THATHUKAL

S.No	EZHU UDAL THATHUKAL	NUMBER OF CASES	PERCENTAGE (%)
1	Saaram	20	100%
2	Senneer	6	30%
3	Oon	0	0%
4	Kozhupu	6	30%
5	Enbu	6	30%
6	Moolai	0	0%
7	Sukkilam /Suronitham	0	0%

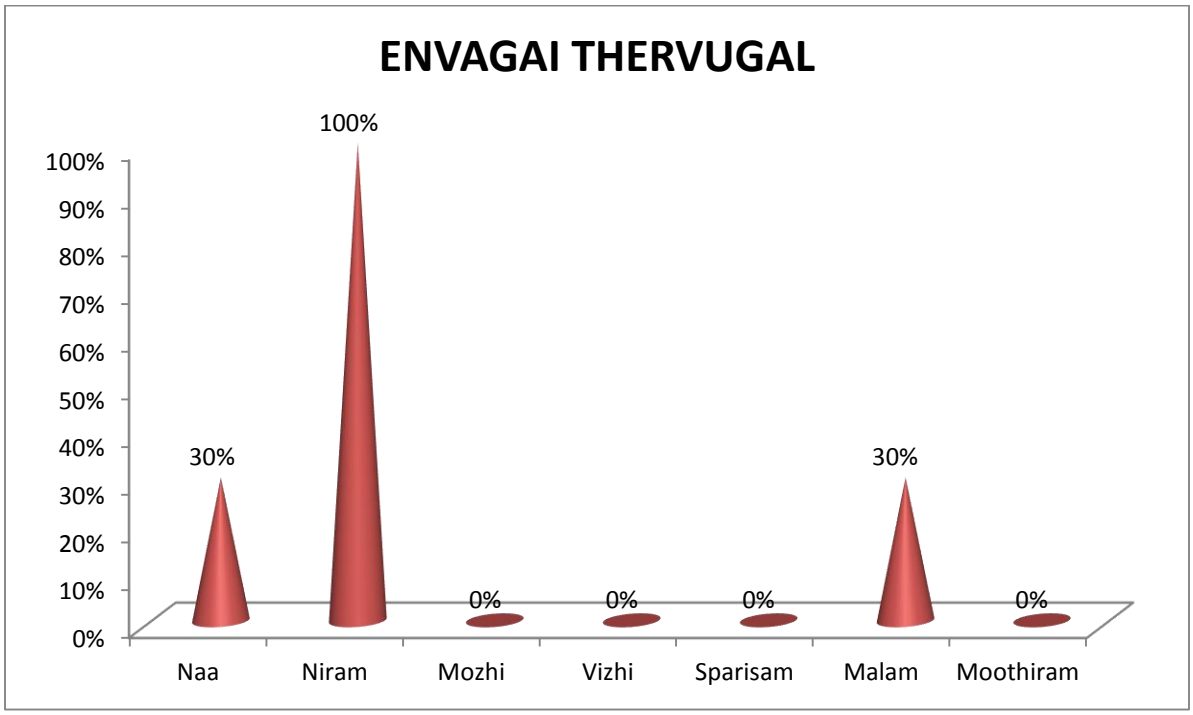


INFERENCE

Out of 20 patients, Saaram was affected in 20 patients (100%), Senneer was affected in 6 patients (30%),Kozhupu was affected in 6 patient (30%) ,Enbuwas affected in 6 patients (30%).

EN VAGAI THERVUGAL

S.No	EN VAGAI THERVUGAL	NUMBER OF CASES	PERCENTAGE (%)
1	Naa	6	30%
2	Niram	20	100%
3	Mozhi	0	0%
4	Vizhi	0	0%
5	Sparisam	0	0%
6	Malam	6	30%
7	Moothiram	0	0%

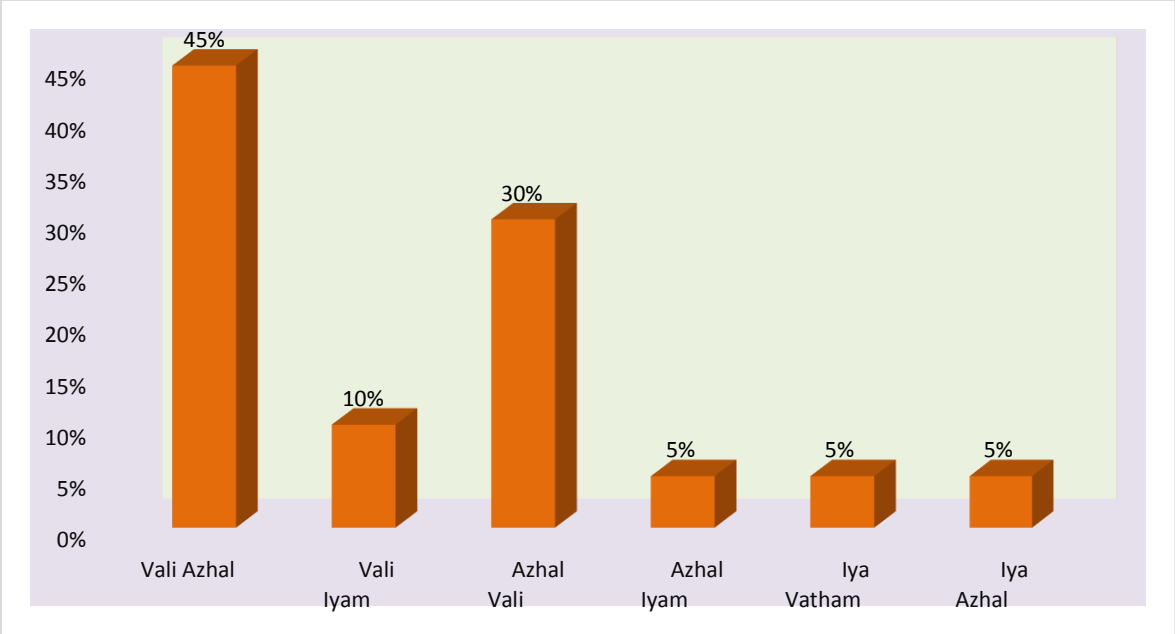


INFERENCE

In Envagaithervu, Naa was affected in 6 patients (30%), Niram was affected in 20 patients (100%), Vizhi was affected in 0 patients (0%) and Malam was affected in 6 patient (30%)

NAADI

S.No	NAADI	NUMBER OF CASES	PERCENTAGE (%)
1	ValiAzhal	9	45%
2	ValiIyam	2	10%
3	AzhalVali	6	30%
4	AzhalIyam	1	5%
5	IyaVatham	1	5%
6	Iya Azhal	1	5%

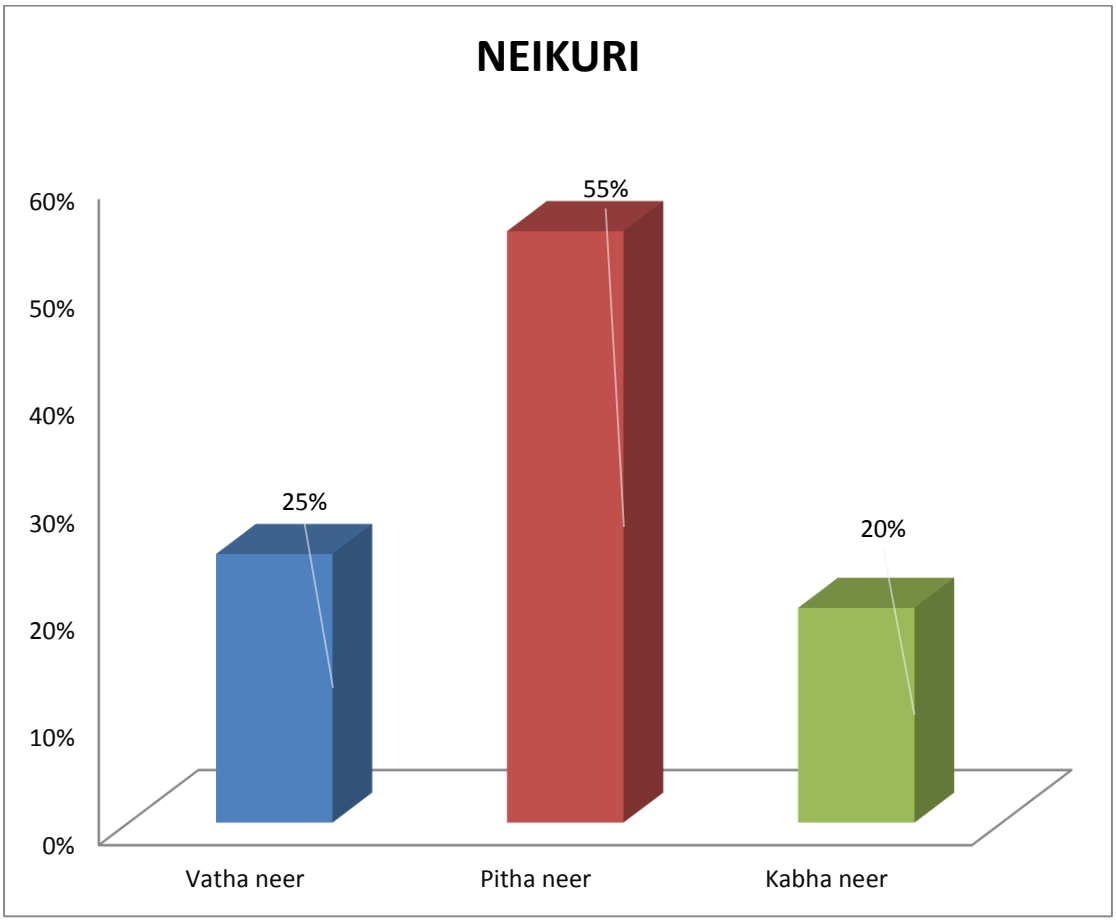


INFERENCE

9 patients (45%) had Valiazhal naadi, 2 patients (10%) had ValiIyam naadi, 6 patients (30%) had Azhalvali naadi, 1 Patient (5%) had Azhaliyam naadi,1 Patient (5%) had Iyavatham naadi, 1 patient (5%) had Iya azhal naadi .

NEIKURI

S.No	THATHU	NEIKURI	NUMBER OF CASES	PERCENTAGE (%)
1	Vathaneer	Spread like snake	5	25%
2	Pithaneer	Spread like ring	11	55%
3	Kabhaneer	Spread like pearl	4	20%

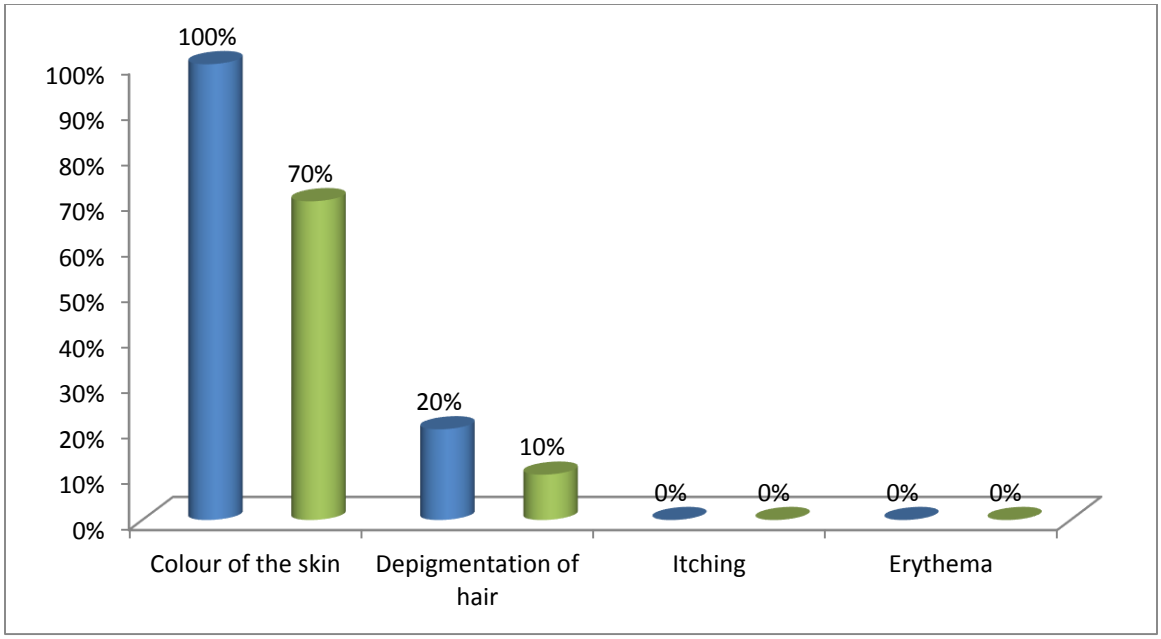


INFERENCE

5 patients (25%) had vathaneer, 11 patients (55%) had Pithaneer, and 4 patients (20%) had Kabhaneer.

CLINICAL FEATURES

SIGN AND SYMPTOMS	BEFORE TREATMENT		AFTER TREATMENT	
	NO.OF	PERCENTAGE	NO.OF CASES	PERCENTAGE
Colour of skin	20	100%	14	70%
Depigmention of hair	3	15%	2	10%
Itching	0	0%	0	0%
Erythema	0	0%	0	0%



INFERENCE :

According to the clinical features, Out of 20 patients ,20 patient had colour changes in skin before treatment , After treatment 14 patient had recovery of skin color changes, 3 patient had depigmentation of hair before treatment ,After treatment 2 patient had recovery of the depigmented hair .

HAEMOGLOBIN LEVEL

S.no	Op. no	Patient's name	Age/Sex	Before treatment	After treatment
1	533	Mrs.Hemavathy	49/F	10 gm	11 gm
2	547	Mr.Naveenkumar	33/M	11.5 gm	12 gm
3	780	Mrs. Selvi	30/F	9.4gm	10.3 gm
4	2706	Mr.Pazhanivel	39/M	9.8 gm	10.4 gm
5	3110	Mr. Selvaraj	39/M	13 gm	13 gm
6	6245	Mr.Vinoth	25/M	11 gm	12.5 gm
7	6408	Mr.Srinivasan	49/M	13 gm	13 gm
8	4028	Mrs. Selvi	31/F	10.2 gm	11.5 gm
9	627	Mrs. Shanthi	33/F	10.5 gm	12 gm
10	783	Mr.Gunasekaran	39/M	9gm	12gm
11	897	Mrs.Prema	45/F	10.4 gm	11.6gm
12	3029	Mrs.Anjalai	47/F	9gm	10.4gm
13	3061	Mis. Uma	25/F	9.8gm	12.4 gm
14	5471	Mr. Mirath	24/M	10.6 gm	11.3 gm
15	1927	Mis.Seetha	23/F	9.9 gm	10.6 gm
16	6847	Mr.Periyasamy	45/M	10gm	10.8gm
17	6893	Mr.Manikandan	19/M	11gm	11.2gm
18	3074	Mrs. Uma	45/ F	9 gm	12 gm
19	7153	Mr.Appalnaidu	38/ M	12 gm	13 gm
20	8527	Mr.Ramamurthi	32/M	11 gm	12.3 gm

LABORATORY INVESTIGATION

HAEMATOLOGY REPORT

S.No	OP.No	Name	Age/Sex	Before Treatment							DC %	After Treatment			Urine Analysis Before Treatment			Urine Analysis After Treatment			
				TC cells/ cu.mm	DC %			ESR		TC Cells/cu. mm				ESR		Albumin	sugar	Deposit	Albumin	sugar	deposit
					P	L	E	½ hr	1 hr		P	L	E	½ hr	1 hr						
1	533	Mrs.Hemavathy	49/F	10700	63	34	3	11	24	10700	62	33	5	8	14	NIL	NIL	NIL	NIL	NIL	NIL
2	547	Mr.Naveenkumar	33/M	9400	57	38	5	9	15	9500	60	36	4	8	10	NIL	NIL	NIL	NIL	NIL	NIL
3	780	Mrs. Selvi	30/F	7600	48	46	6	8	14	7800	52	43	5	8	10	NIL	+	Opc	NIL	NIL	NIL
4	2706	Mr.Pazhanivel	39/M	9900	53	40	7	15	30	10200	57	37	6	10	18	NIL	NIL	NIL	NIL	NIL	NIL
5	3110	Mr. Selvaraj	39/M	7200	54	40	6	10	20	7800	58	37	5	10	18	NIL	N IL	NIL	NIL	NIL	NIL
6	6245	Mr.Vinoth	25/M	9700	58	35	7	18	35	10000	62	33	5	15	30	NIL	NIL	Opc	NIL	NIL	NIL
7	6408	Mr.Srinivasan	49/M	8600	55	39	6	5	45	8800	57	37	6	15	30	+	NIL	NIL	NIL	NIL	NIL
8	4028	Mrs. selvi	31/F	9200	61	35	4	33	60	9300	63	34	3	17	30	NIL	NIL	NIL	NIL	NIL	NIL
9	627	Mrs. Shanthi	33/F	9000	54	40	6	30	62	9200	58	37	5	15	30	NIL	++	oec	NIL	NIL	NIL
10	783	Mr.Gunasekaran	39/M	8000	55	39	6	12	30	8100	57	37	6	10	15	NIL	NIL	NIL	NIL	NIL	NIL
11	897	Mrs. Prema	45/F	8000	55	41	4	8	14	8600	58	39	3	8	10	NIL	NIL	opc	NIL	NIL	NIL
12	3029	Mrs. Anjalai	47/F	7400	50	46	4	9	15	7800	58	40	2	8	14	+	NIL	opc	NIL	NIL	NIL
13	3061	Mis.Uma	25/F	10000	62	34	4	10	20	10100	58	35	7	10	18	NIL	NIL	NIL	NIL	NIL	NIL
14	5471	Mr. Mirath	24/M	8200	63	33	4	15	30	8600	67	30	3	10	15	NIL	NIL	NIL	NIL	NIL	NIL
15	1927	Mis. Seetha	23/F	7800	56	37	7	33	60	7900	58	40	2	17	30	NIL	NIL	Oec	NIL	NIL	NIL
16	6847	Mr.periyasamy	45/M	9700	58	39	3	18	35	9900	62	36	2	15	30	NIL	NIL	NIL	NIL	NIL	NIL
17	6893	Mr.Manikandan	19/M	9200	57	36	7	8	14	9600	60	34	6	5	10	+	NIL	NIL	NIL	NIL	NIL
18	3074	Mrs. Uma	45/F	10000	62	34	4	10	20	10100	58	35	7	10	18	NIL	NIL	opc	NIL	NIL	NIL
19	7153	Mr.Appalnaidu	38/M	7900	64	33	3	10	20	8200	68	30	2	8	10	NIL	+++	NIL	NIL	NIL	NIL
20	8527	Mr.Ramamurthi	32/M	8600	49	46	5	5	45	8800	56	40	4	15	30	NIL	NIL	NIL	NIL	NIL	NIL

TC – Total count, DC – Differential count, P – Polymorphs, L – Lymphocyte, E – Eosinophil, ESR – Erythrocyte Sedimentation Rate, Oec – Occasional epithelial cells, Opc – Occasional pus cells, Alb – Albumin, Sug – Sugar, Dep – Deposit

LIST OF PATIENTS

S.no	Op. no	Patient's name	Age/Sex	Occupation	Date of medicine
1	533	Mrs.Hemavathy	49/F	House wife	05-10-2015
2	547	Mr.Naveenkumar	33/M	Office worker	05-10-2015
3	780	Mrs. Selvi	30/F	House wife	05-10-2015
4	2706	Mr.Pazhanivel	39/M	Business	12-10-2015
5	3110	Mr. Selvaraj	39/M	Business	20-11-2015
6	6245	Mr.Vinoth	25/M	Office worker	01-12-2015
7	6408	Mr.Srinivasan	49/M	Farmer	12-12-2015
8	4028	Mrs. Selvi	31/F	House wife	28-12-2015
9	627	Mrs. Shanthi	33/F	House wife	04-01-2016
10	783	Mr.Gunasekaran	39/M	Farmer	04-01-2016
11	897	Mrs.Prema	45/F	House wife	04-01-2016
12	3029	Mrs.Anjalai	47/F	House wife	11-01-2016
13	3061	Mis. Uma	25/F	Student	11-01-2016
14	5471	Mr. Mirath	24/M	Office	21-01-2016
15	1927	Mis.Seetha	23/F	Shop	08-02-2016
16	6847	Mr.Periyasamy	45/ M	Farmer	22-02-2016
17	6893	Mr.Manikandan	19/M	Student	22-02-2016
18	3074	Mrs. Uma	45/ F	House wife	11-02-2016
19	7153	Mr.Appalnaidu	38/ M	Shop	24-03-2016
20	8527	Mr.Ramamurthi	32/M	Auto Driver	29-03-2016

VASI SCORE

S. NO	OP. NO	HEAD				UPPER LIMB				TRUNK				LOWER LIMB				TOTAL SCORE	
		D_H		A_H		D_H		A_H		D_T		A_T		D_L		A_L			
		BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT
1	5471	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0.1	0.01
2	3061	2	1	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0.4	0.1
3	1927	2	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0.6	0
4	897	3	1	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0.6	0.1
5	533	0	0	0	0	3	1	5	3	0	0	0	0	0	0	0	0	3	0.6
6	780	0	0	0	0	2	1	4	2	0	0	0	0	0	0	0	0	1.6	0.4
7	8527	0	0	0	0	2	1	2	1	0	0	0	0	0	0	0	0	0.8	0.2
8	7153	0	0	0	0	2	1	2	1	0	0	0	0	0	0	0	0	0.8	0.2
9	3074	0	0	0	0	2	1	2	1	0	0	0	0	0	0	0	0	0.8	0.2
10	627	0	0	0	0	2	1	2	1	0	0	0	0	0	0	0	0	0.8	0.1
11	3029	0	0	0	0	3	2	2	1	0	0	0	0	0	0	0	0	1.2	0.4
12	4028	0	0	0	0	0	0	0	0	2	0	4	0	0	0	0	0	2.4	0
13	547	0	0	0	0	0	0	0	0	2	1	3	1	0	0	0	0	1.8	0.3
14	6893	0	0	0	0	0	0	0	0	1	1	1	1	0	0	0	0	0.3	0.03
15	2706	0	0	0	0	0	0	0	0	0	0	0	0	3	0	1	0	1.2	0
16	3110	0	0	0	0	0	0	0	0	0	0	0	0	3	1	3	2	3.6	0.8
17	783	0	0	0	0	0	0	0	0	0	0	0	0	2	1	2	1	1.6	0.4
18	6245	0	0	0	0	0	0	0	0	0	0	0	0	2	1	2	1	1.6	0.4
19	6847	0	0	0	0	0	0	0	0	0	0	0	0	3	2	2	1	2.4	0.8
20	6408	0	0	0	0	0	0	0	0	0	0	0	0	2	1	3	2	2.4	0.8

CASE REPORT FOR PATIENTS BASED ON VASI SCORE

S. NO	OP. NO	AGE/ SEX	DOA	DOD	INITIAL VASI SCORE	POST VASI SCORE	RESULT
1	5471	24/M	21/01/2016	08/03/2016	0.1	0.01	Mild
2	3061	25/F	11/01/2016	27/02/2016	0.4	0.1	Marked
3	1927	23/F	08/02/2016	26/03/2016	0.6	0	Marked
4	897	45/F	04/01/2016	20/02/2016	0.6	0.1	Marked
5	533	49/F	05/10/2015	21/11/2015	3	0.6	Moderate
6	780	30/F	05/10/2015	21/11/2015	1.6	0.4	Moderate
7	8527	32/M	29/03/2016	15/05/2016	0.8	0.2	Moderate
8	7153	38/M	24/03/2016	09/05/2016	0.8	0.2	Moderate
9	3074	45/F	11/02/2016	29/03/2016	0.8	0.2	Moderate
10	627	33/F	04/01/2016	20/02/2016	0.8	0.1	Moderate
11	3029	47/F	11/01/2016	27/02/2016	1.2	0.4	Moderate
12	4028	31/M	28/12/2015	07/02/2016	2.4	0	Marked
13	547	33/M	05/10/2015	21/11/2015	1.8	0.3	Moderate
14	6893	19/M	21/01/2016	08/03/2016	0.3	0.03	Mild
15	2706	39/M	12/10/2015	28/11/2015	1.2	0	Marked
16	3110	39/M	20/11/2015	06/01/2016	3.6	0.8	Moderate
17	783	39/M	04/01/2016	20/02/2016	1.6	0.4	Moderate
18	6245	25/M	01/12/2015	17/01/2016	1.6	0.4	Moderate
19	6847	45/M	22/02/2016	09/04/2016	2.4	0.8	Marked
20	6408	49/M	12/12/2015	29/01/201	2.4	0.8	Marked

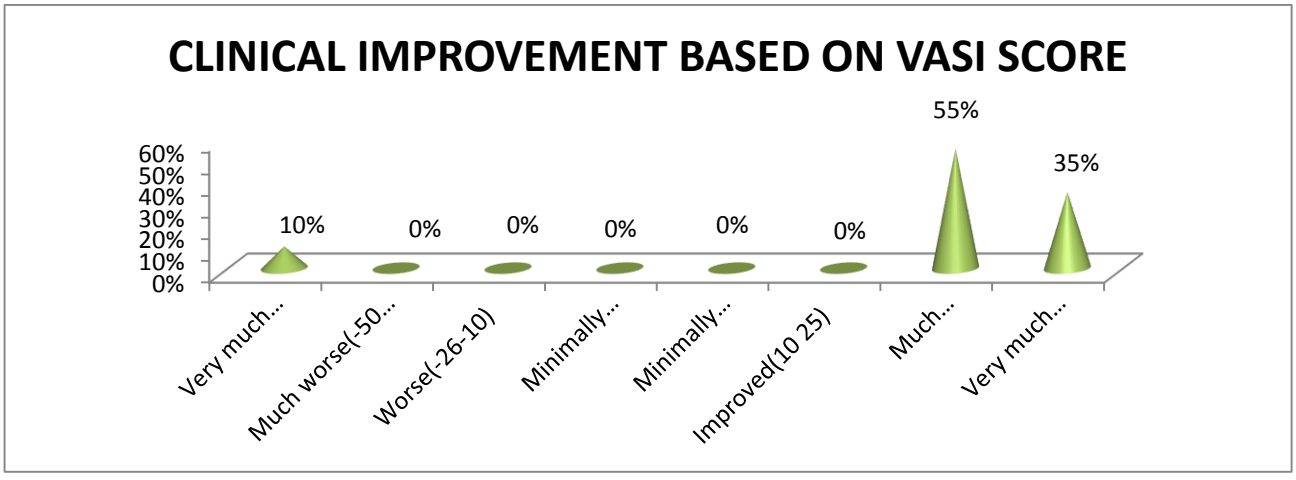
FORMULA FOR VASI SCORE:

VA SI SCORE= 0.1 × (D_H) A_H + 0.2 × (D_U) A_U + 0.3 × (D_T) A_T + 0.4 × (D_L) A_L.

CLINICAL IMPROVEMENT BASED ON VASI SCORE:

PATIENT TREATED WITH BOTH INTERNEL& EXTERNEL MEDICINE:

S.NO	EFFECT OF TRIAL MEDICINE	NO OF PATIENTS	PERCENTAGE
1	Very much worse(~-50)	2	10%
2	Much worse(-50 -25)	0	0%
3	Worse(-26-10)	0	0%
4	Minimally Worse(-10 0)	0	0%
5	Minimally Improved(0 10)	0	0%
6	Improved(10 25)	0	0%
7	Much Improved(25 50)	11	55%
8	Very much Improved(+50~)	7	35%

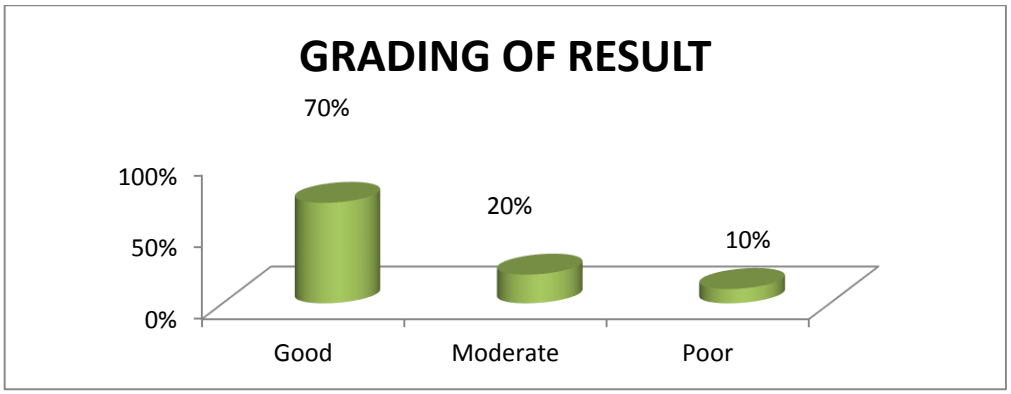


INFERENCE:

Among 20 patients treated with internal and external medicine in OPD, 2 out of 20 patient achieved very much worse condition(10%) in VASI score,11 out of 20 patient achieved much improved (55%) in VASI score,7 patient achieved very much improved (35%) in vasi score.

GRADING OF RESULTS

S.No	GRADING	NUMBER OF CASES	PERCENTAGE (%)
1	Good	14	70%
2	Moderate	4	20%
3	Poor	2	10%



INFERENCE :

Out of 20 patients,14 patient shows good result (70%),4 patient shows moderate result (20%), and 2 patient shows poor result (10%).

OP.NO:6408

BEFORE TREATMENT

DURING TREATMENT

AFTER TREATMENT

OP.NO:1927

BEFORE TREATMENT

DURING TREATMENT

AFTER TREATMENT

OP.NO:547

BEFORE TREATMENT

DURING TREATMENT

AFTER TREATMENT

DISCUSSION

DISCUSSION

Venpulli is an acquired idiopathic depigmentory condition and is characterized by completely depigmented macules and patches of varying sizes and shapes.

The patients were examined based on Siddha and as well as modern aspects. All the necessary investigations were made during the study. The results obtained from their studies were discussed below for better conclusion.

The clinical study of venpulli was carried out in post graduate department of Maruthuvan Govt. Siddha medical College, attached to Arignar Anna Hospital of Indian Medicine, Arumbakkam, and Chennai – 106 during 2014-2016. 20 Patients of both sexes of various adult age groups satisfying the inclusion and exclusion criteria were selected. All necessary investigation was done for them and photographs of the lesion were taken. A day before starting the trial treatment, cleaning of mukkutram by purgation will be carried out by Agasthiyar Kuzhambu-100mg, early morning with hot water. All the patients were treated with trial medicine. Hence with the help of trial medicines results and observation are noted for this study.

Trial medicine administered was **Rasacheenee Chooranam (internal)** – 10mg bid after food

Vehicle: palm jaggery

Duration: 48 days (1 Mandalam).

Karkadagasingi pattru for external medicine.

1. Sex distribution:

Among 20 cases 11 were males and 9 were females.

2. Age distribution:

Majority of the case that is 40% were in the 3rddecade, 30% were in the 4th decade, 25% were in the 2nd decade, 5% were in the 1stdecade

3. Occupation :

Mixed categories of people are affected, from housewife to office worker, students to retired person. House wifes and office workers were affected commonly.

4. Food habits:

Among 20 patients, 6 patients eat vegetarian food and others eat mixed diet.

5. According to season:

The highest incidence were noted in Munpani Kaalam (50%) and 20% were noted in koothir Kaalam, 15% were noted in kaarkalam and pinpani kaalam

6. Distribution of thinai:

According to thinai, the highest distribution 77.5% was noted in neithal, 12.5% in Marutham and 10% noted in kurinji.

7. On clinical manifestations:

All of my patients had skin colour changes, 15% patient had depigmentation of hair.

8. Mukkutram:

Distribution of vatham:

Among the patients 100% were affected in Viyanan, 30% patient were affected in Pranan and Abanan, 15% patient were affected in koorman.

a. Affected pranan produced dyspnoea.
b. Affected Abanan produced constipation.
c. Affected viyanan produce difficulty in movemen & skin colour changes.
d. Affected Koorman produced impairment of eye sight

Distribution of pitham:

Among the treated patients 100% were affected in Pirasagam, 30% were affected in Ranjagam, and 35% were affected in alosagam.

1. Affected Ranjagam produced pallor of skin, eye and reduced hemoglobin.
2. Affected pirasagam produced hypopigmentation of skin.
3. Affected Alosagam produced impairment of eye sight.

Distribution of Kabham:

Among the patients, 30% were affected in Avalambagam and 30% were affected in Santhigam.

1. Affected Avalambagam produced dyspnoea.
2. Affected Santhigam produced low back pain, knee joint pain.

9. Ezhu Udal Thathukkal:

Among the treated patients saram was affected in 100% of patients, senneer in 30% ,kozhupu and enbu in 30%

1. Saaram produce tiredness
2. Senner produce decreased level of haemoglobin.
3. Kozhupu and enbu produce restricted movements in both knee joints.

10. Enn vagai thervugal:

In this, Naa was affected in 30%, Niram in 100%, Malam in 30%

1. Naa was affected cases results in paleness of the tongue.
2. Niram was affected all cases results in the colour of skin changed into white.

11. Naadi:

On examination of naadi, 45% had vazhi azhal naadi, 10% had vali iyam naadi, 30% had Azhalvali naadi, 5% had Azhal iyam naadi, 5% had kabha pitha naadi and 5% had kabha vatha naadi.

12. Neikuri:

On Neikuri examination 25% were having vatha neer, 55% were having pitha neer and 20% were having kabha neer.

A drop of gingely oil dropped into the early morning urine sample in a bowl may result in spread like snake called vatham, like a ring in pitham, like pearl in kabham.

1.Mode of action of the drug:

According to suvai:

The trial medicine **Rasacheenee chooranam** has bitter and astringent taste.

This taste will equalize the increased pitham which is the main cause for Venpulli. This medicine acts against the increased pitham. So it is considered as Ethirurai maruthuvam.

Karkadagasingi pattru for external

According to veeriyam: (nature)

The trial medicine Rasacheenee chooranam possesses thatpa veeriyam. So it cures pitha diseases.

By this Rasacheenee chooranam treats Venpulli.

1. Bio chemical analysis:

Rasacheenee chooranam has starch, Iron,zinc ,calcium,reducing sugar and starch.

2. Toxicological analysis:

Acute, Sub acute and sub chronic toxicity studies were conducted at Baid Methaw college of Pharmacy. At the end of toxicity studies the hematological parameters (TC, DC and Hb), Biochemical parameters (LFT, KFT) and histopathology of vital organs like Liver, Kidney, Spleen and Lungs were carried out. Rasacheenee Chooranam shows no toxic effect.

3. Statistical analysis:

The preclinical studies of trial medicine Rasacheenee Chooranam statistically analysed and showed significant result.

5.Results after treatment:

Many of the patients were relieved of their problem, 70% showed good result , 30% Showed moderate result ,who are relieved of their problem, 10% showed poor result were not relieved of their problem.

SUMMARY

SUMMARY

- I like to summarize this study with the following highlights.
- Females are more prone to get than males according to my studies.
- In age distribution, 3rd and 4th decades of people are more affected.
- House wifes and office workers occupy the first two places in occupational classification.
- Most of the patient had mixed diet.
- Higher incidence of cases were noted in munpani kalam (Mid Dec – Mid Feb)
- Etiology of venpulli patient had unknown causes in my studies.
- In the disturbance of vatham**,**Among the patients 100% were affected in Viyanan 30% patient were affected in Pranan andAbanan,15%patient were affected in koorman.
- In the disturbance of Ezhu udal thathukkal, 100% were affected by saaram, 30% were affected by senneer and 30% were affected by enbu and kozhupu
- In Naadi, most of the patients having vali azhal naadi(45%)..
- In Neikuri examination 55% were having pitha neer.
- Among the patients, 100% had depigmentation of skin colour, 15% had depigmentation of hair.
- All of my patients were administered with my trial medicine **Rasacheenee chooranam** –10mgs bd with Palm jaggery after food for a period of 48 days.- Internal medicine.
- External medicine –**Karkadagasingi pattru.**
- After treatment with this trial medicine, shows complete cure of venpulli in 35% patient, and give moderate result in (55%).

CONCLUSION

CONCLUSION

- Vitiligo is the most common depigmentary disorder it may occur due to various causes and it leads to mental stress and strain. Hence it's one of the causes of psychomatic disorder.
- Venpulli (vitiligo) is mainly due to derangements of piththa humour.
- The trial medicine,Rasacheenee chooranam for internal and karkadagasingi pattru for external.
- The internal medicine of Rasacheenee chooranam had kaippu and thuvarppu suvai, neutralize the increased piththam thereby it act as ethururai maruthuvam.
- The Rasacheenee chooranam reveals no toxicity in the preclinical studies and hence proved to be safe for human administration.
- No contra indication was reported during the course of the treatment
- The trial medicine gave good result for skin colour changes in patients.
- The preparation of trial medicine is economical.
- The rasacheenee chooranam does not produce any toxicity in preclinically. So it is nontoxic and safe drug for venpulli.
- From this clinical studies, I conclude that the trial medicines which gives a 65-70% of improvement within minimum of 48 days. Rasacheenee chooranam gave complete recovery of venpulli in 35% patient.
- Therefore the author concluded that the trial medicine **RASACHEENEE CHOORANAM (INTERNAL)** and **KARKADAGASINGI PATTRU (EXTERNAL)** can give better solutions for venpulli (vitiligo).

ANNEXURE – I

CERTIFICATES

The Tamil Nadu Dr.M.G.R. Medical University

#69, Anna salai, Guindy, Chennai-600 032.

This certificate is awarded to

Dr./~~Mr./Ms.~~ NANDHINI . R

for participating as ~~Resource Person~~ / Delegate in the Fourteenth Workshop on

"Research Methodology & Biostatistics"

for AYUSH Post Graduates & Researchers

Organised by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University from 5th to 9th May 2014.

Dr. N. KABILAN M.D. (Siddha)
Reader, Dept. of Siddha

Dr. JHANSI CHARLES, M.D.
Registrar

Prof. Dr. D. SHANTHARAM, M.D., D.Diab.,
Vice-Chancellor

சித்த மருத்துவ மைய அராய்ச்சி நிலையம், சென்னை — 600 106
सिद्ध केंद्रीय अनुसन्धान संस्थान, अण्णा सरकारी अस्पताल परिसर, अरुम्बाक्कम, चेन्नई - 600106

SIDDHA CENTRAL RESEACH INSTITUTE

(Central Council for Research in Siddha, Ministry of AYUSH, Govt. of India)
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14.06.2016

Name of the student: by Dr. R. Nandhini, III year MD Student,

Department of Pothu Maruthuvam, Government Siddha Medical College, Chennai-600 106

PHYSICO-CHEMICAL ANALYSIS OF RASACHEENEE CHOORANAM

S.No	Physicochemical Parameter		Mean
1.	Loss on Drying at 105°C	:	10.52 %
2.	Total Ash	:	2.95 %
3.	Water soluble Ash	:	1.27 %
4.	Acid insoluble Ash	:	0.58 %
5.	Water Soluble Extractive	:	4.0 %
6.	Alcohol Soluble Extractive	:	2.95 %
7.	pH	:	5.5
TLC/HPTLC		:	Annexed

(R. Shakila)
Research Officer (Chemistry)

16/6/16

(Dr. P. Sathiyarajeswaran)
Assistant Director (Scientist 2) I/c

Dr. P. SATHIYARAJESWARAN
Assistant Director (Scientist-2) 1/C
Siddha Central Research Institute (CCRS)
Min. of AYUSH, Govt. Of India
Arumbakkam, Chennai-600 106.

Certificate

This is certify that the project titled Toxicological activity of RASACHEENEE CHOORANAM in rats has been approved by the

IAEC No: IAEC/XLIV/14/CLBMCP/2014

Name of Chairman/ Member Secretary IAEC:

08/09/14

Signature with date

Bapd Medha College of Pharmacy CHENNAI-97

C.L.BAID METHA COLLEGE OF PHARMACY

(An ISO 9001-2000 certified institute)

Jyothi Nagar, Old Mahabalipuram Road

Thoraipakkam, Chennai – 600 097

CERTIFICATE

This is to certify that the project entitled, **Toxicological and Pharmacological study on RASACHEENEE CHOORANAM** in rats submitted in partial fulfilment for the degree of **M.D. (siddha)** was carried out at C.L. Baid Metha college of Pharmacy, Chennai-97, in the Department of Pharmacology during the academic year of 2014-2015.

(Dr.P.Muralidharan)

Mr. P.Muralidharan, M.Pharm, Ph.D
Professor and Head
Department of Pharmacology,
C.L.Baid Metha college of pharmacy,
Chennai-97

சித்த மருத்துவ மைய ஆராய்ச்சி நிலையம், அரும்பாக்கம், சென்னை - 600 106
सिध्द केन्द्रीय अनुसंधान संस्थान, अरुम्बाक्कम, चेन्नई- 600 106
Siddha Central Research Institute
Arignar Anna Govt. Hospital Campus, Arumbakkam, Chennai-600 106
(Central Council for Research in Siddha, Department of AYUSH,
Ministry of Health & Family Welfare, Govt. of India)
Phone: 044-26214925, Tele Fax: 044-26214809, E.mail: crisiddha@gmail.com, Web: www.crisiddha.tn.nic.in

22nd February 2016

CERTIFICATE

Certified that the drugs submitted for identification by Dr. R. Nandhini, PG III year, Department of Pothu maruthuvam, Government Siddha Medical College, Arumbakkam, Chennai - 600 106, are identified as

1. Etti - *Strychnos nux – vomica* L. (Seed)
2. Serankottai - *Semecarpus anacardium* L. (Fruit)
3. Parankippattai - *Smilax china* L. (Tuberous root)
4. Karkadagasringi - *Pistacia chinensis* Bunge (Gall)
5. Kodiveli verpattai - *Plumbago zeylanica* L. (Rootbark)

Sasikala Ethirajulu

Sasikala Ethirajulu
Consultant (Pharmacognosy)

22/2/16

P.Sathiyarajeswaran
Assistant Director Incharge

Dr. P. SATHIYARAJESWARAN
Assistant Director (Scientist-2) 1/C
Siddha Central Research Institute (CCRS)
Min. of AYUSH, Govt. Of India
Arumbakkam, Chennai-600 106.

சித்த மருத்துவ மைய ஆராய்ச்சி நிலையம், அரும்பாக்கம், சென்னை - 600106
सिद्ध केन्द्रीय अनुसंधान संस्थान, अरुम्बाक्कम, चेन्नै - 600106
Siddha Central Research Institute
(Central Council for Research in Siddha, Ministry of AYUSH, Govt. of India)
Arumbakkam, Chennai – 600106
[Ph: 044-26214925, 26214809, Fax: 26214809, Email: crisiddha@gmail.com, Web: www.siddhacouncil.com]

15.3.2016

CERTIFICATE

Certified that the samples submitted for identification by Dr. R. Nandini, III year MD Student, Department of Pothu Maruthuvam, Government Siddha Medical College, Chennai-600 106 are identified as Rasam – Mercury, Gandhagam – Sulphur and Thalagam - Arsenic trisulphide.

(R. Shakila)
Research Officer (Chemistry)

P. Elankani 15/03/16
(Dr. P.Elankani)
for Research Officer (Scientist 2)-I/c

GOVERNMENT SIDDHA MEDICAL COLLEGE
Arumbakkam, Chennai-106

Communication Of The Decision Of Institutional Ethical Committee (IEC)

IEC No: GSMC-CH-ME-3/005/2014

Protocol title: AN OPEN CLINICAL STUDY ON VENPULLI (VITILIGO) WITH THE EVALUATION OF SIDDHA DRUG RASACHEENEE CHOORANAM(INT)KARKADAGASINGI PRTTRU(EXT).
Principal Investigator: DR. R.NANDHINI
Name & Address of Institution: Government Siddha Medical College, Arumbakkam, Chennai-106
☐ New Review ☐ Revised Review ☐ Expedited Review
Date of review (DD/MM/YY): 13-06-2014 Date of Previous Review, If Revised Application:
Decision of the IEC ☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions / Reasons / Remarks: (i) Do phase I skin sensibility test for five healthy individuals before giving to patients (ii) Do 90 days repeated oral toxicity study instead of pharmacological activity.
Recommended for a period of 1 year from date of completion of preclinical studies :

Please Note:

- Inform IEC immediately in case of any adverse events/serious drug reaction.
- Seek IECapproval in case of any change in the study procedure, site and investigator
- This approval is valid only for period mentioned above
- IEC member have the right to review the trial with prior intimation.

13/6/14

Dr.P.Jeyaprakashnarayanan
Chairman

Dr. V.Banumathi
Member Secretary

INSTITUTIONAL ETHICAL COMMITTEE

Date:

Sub: IEC review of research proposals.

Ref: Your letter dated

MEMBERS	PARTICIPATION	SIGNATURE
DR.P.JEYAPRAKASH NARAYANAN M.D(S)., Chairman	☑	
DR.V.BANUMATHI M.D(S)., Member Secretary	☐	
DR.N.KABILAN M.D(S)., Clinician- Siddha	☑	13/6/14
DR.P.SATHIYA RAJESWARAN M.D(S)., Clinician- Siddha	☑	
DR.G.AADINATH REDDY,M.Pharm, Ph.D., Pharmacologist	☑	
DR.S.THILAGAVATHY Msc.,Ph.D., Social Scientist	☑	
DR.T.MAHALAKSHMI M.A.,Ph.D., Linguistic Expert	☑	
DR.P.VIDYA M.B.B.S., DMRD., Modern Medicine Expert	☑	
MR.P.SARAVANAN., Public Person	☑	

13/6/14.

Dr.P.Jeyaprakashnarayanan M.D(S).,
Chairman

Dr.V.Banumathi M.D(S).,
Member Secretary

ANNEXURE – II

BIO – CHEMICAL ANALYSIS

BIO-CHEMICAL ANALYSIS OF TRIAL MEDICINE

Preparation of Sodium Carbonate extract:

2 gm of the sample drug is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No	EXPERIMENT	OBSERVATION	INFERENCE
I	**TEST FOR ACID RADICALS**		
1a	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absent
b	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then 2ml barium chloride solution is added.	Absence of White Precipitate	Absent
2	**Test for Chloride:** 2ml of extract is added with dilute nitric acid till the effervescence ceases. Then 2ml of silver nitrate solution is added.	No white precipitate obtained	Absent
3	**Test for Phosphate** 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	NoYellow precipitate obtained	Absent
4	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white precipitate	Absent

5	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentrated Hcl.	Absence of Rotten egg smelling	Absent
6	**Test for Nitrate:** 1gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of reddishbrown gas.	Absent
7a	**Test for Fluoride and oxalate** 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of white precipitate	Absent
b	5 drops of clear solution is added with 2ml of diluted sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganatesolution is added.	No KMNO4 solution Discolourisation obtained	Absent
8	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops of Acetic Acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absent
9	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohol (95%) and introduced into the blue flame.	Absence of Green tinged flame	Absent
II	**TEST FOR BASIC RADICALS**		
10	**Test for lead** 2 ml of the extract is added with 2 ml of Potassium iodide solution.	Absence of Yellow precipitate	Absent
11a	**Test for Copper**	Absence of	Absent

	One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non-luminous part of the flame.	Bluishgreen colouredflame.	
b	2ml of the extract is added with excess of Ammonia solution	Absence of deep blue	Absent
12	**Test for Aluminium** To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess	Absence of White Precipitate.	Absent
13a	**Test for Iron** To the 2 ml of extract, 2 ml of Ammonium Thiocyanate Solution is added.	presence of Blood red colour	Present
b	To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	Blood red colour obtained	Present
14	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	presence of Whiteprecipitate.	Present
15	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	Presence of Whiteprecipitate.	Present
16	**Test for Magnesium** 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	Absence of Whiteprecipitate.	Absent
17	**Test for Ammonium** 2 ml of extract few ml of Nessler's	Absence of Reddish brown	Absent

	Reagent and excess of Sodium Hydroxide solution are added.	precipitate	
18	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Absence of Yellow precipitate	Absent
19	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absent
20	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absence of yellow precipitate	Absent
21	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution.	Absence of Yellow precipitate	Absent
22	**Test for Starch** 2ml of extract is treated with weak iodine solution	presence of Blue colour	Present
23	**Test of reducing Sugar** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	Presence of Green colour	Present
24	**Test of the alkaloids** 2ml of the extract is treated with 2ml of potassium lodide solution.	presence of Red colour	Present
25	**Test of the proteins** 2ml of the extract is treated with 2ml of 5% NaOH, mix well and add 2 drops of copper sulphate solution.	Absence of Violet colour	Absent

RESULTS:

The given sample (Rasacheenee Chooranam) contains

- Calcium
- Zinc
- iron
- reducing sugar
- Alkaloids
- Starch.

ANNEXURE – III

PHYSICO CHEMICAL ANALYSIS

சித்த மருத்துவ மைய ஆராய்ச்சி நிலையம், சென்னை — 600 106
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14.06.2016

Name of the student: by Dr. R. Nandhini, III year MD Student,

Department of Pothu Maruthuvam, Government Siddha Medical College, Chennai-600 106

PHYSICO-CHEMICAL ANALYSIS OF RASACHEENEE CHOORANAM

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5.	Water Soluble Extractive	:	4.0 %
6.	Alcohol Soluble Extractive	:	2.95 %
7.	pH	:	5.5
TLC/HPTLC		:	Annexed

(R. Shakila)
Research Officer (Chemistry)

14/6/16

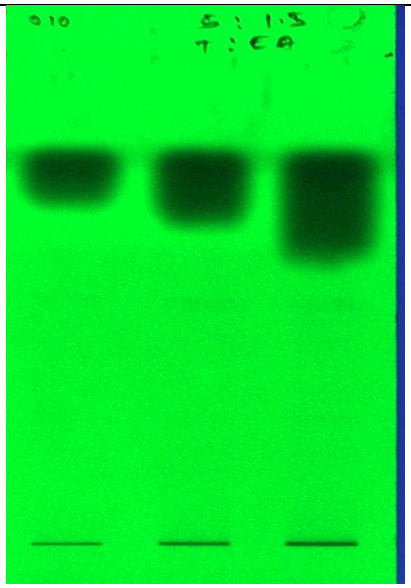
(Dr. P. Sathiyarajeswaran)
Assistant Director (Scientist 2) I/c

Dr. P. SATHIYARAJESWARAN
Assistant Director (Scientist-2) I/C
Siddha Central Research Institute (CCRS)
Min. of AYUSH, Govt. Of India
Arumbakkam, Chennai-600 106.

Sample Name/ID –Rasacheenee chooranam

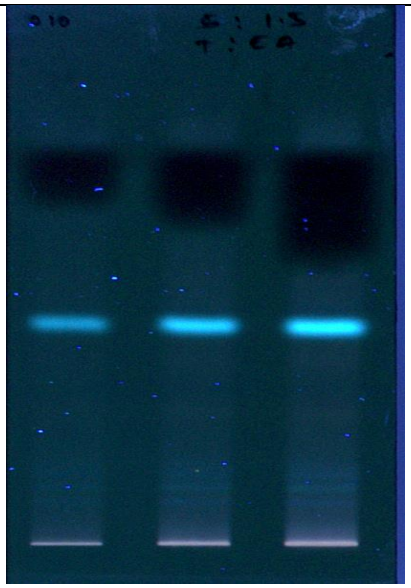
Stationary Phase - Silica Gel 60 F_{254}

Mobile Phase – Toluene : Ethyl Acetate (5 : 1.5 v/v)



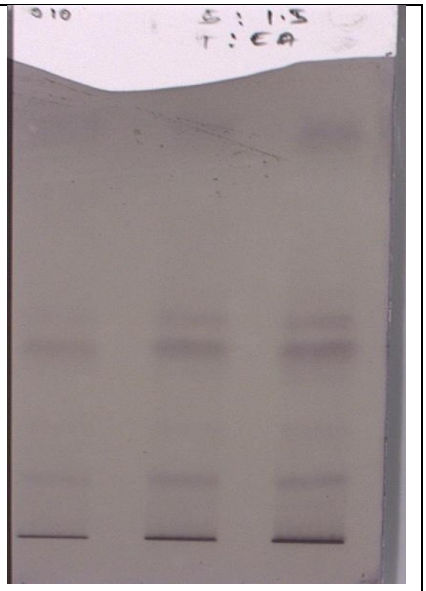
254 nm

Color	R_f value(s)
Green	0.49
Green	0.76



366 nm

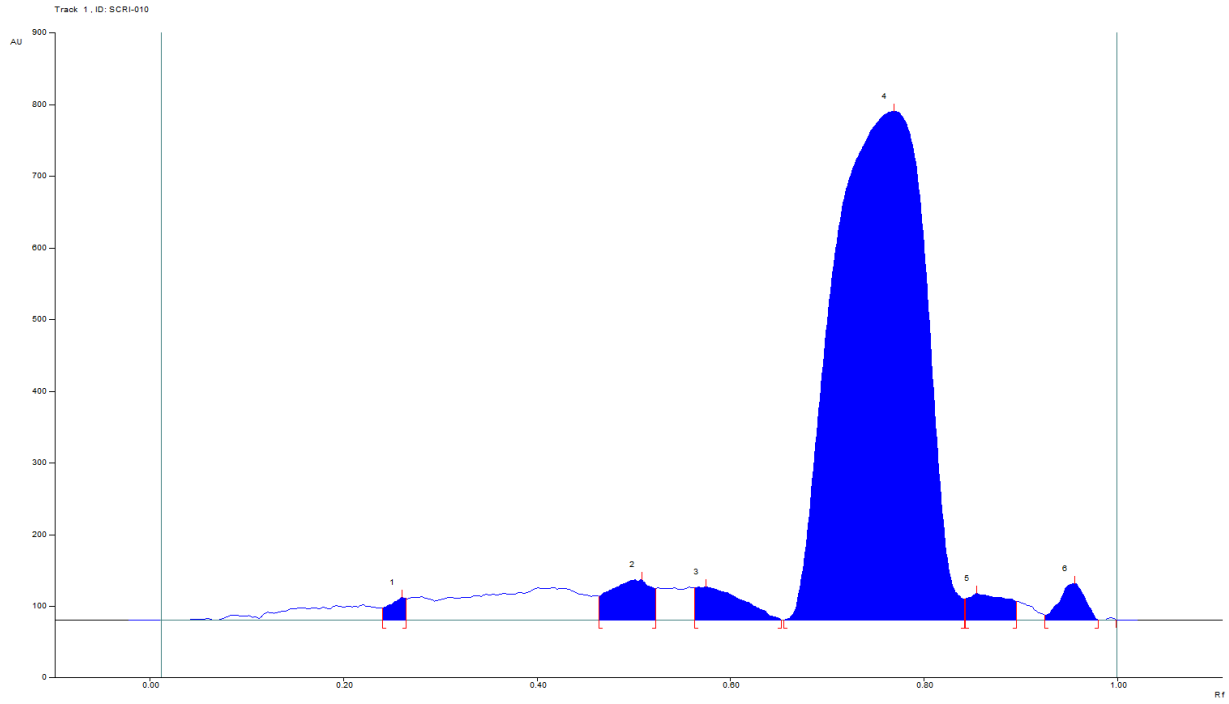
Color	R_f value(s)
Light Green	0.09
Light Green	0.11
Light Blue	0.14
Fluorescent Blue	0.44
Dark Blue	0.76



575 nm (Derivatized)

Color	R_f value(s)
Light purple	0.13
Light purple	0.39
Light purple	0.45
Light purple	0.82

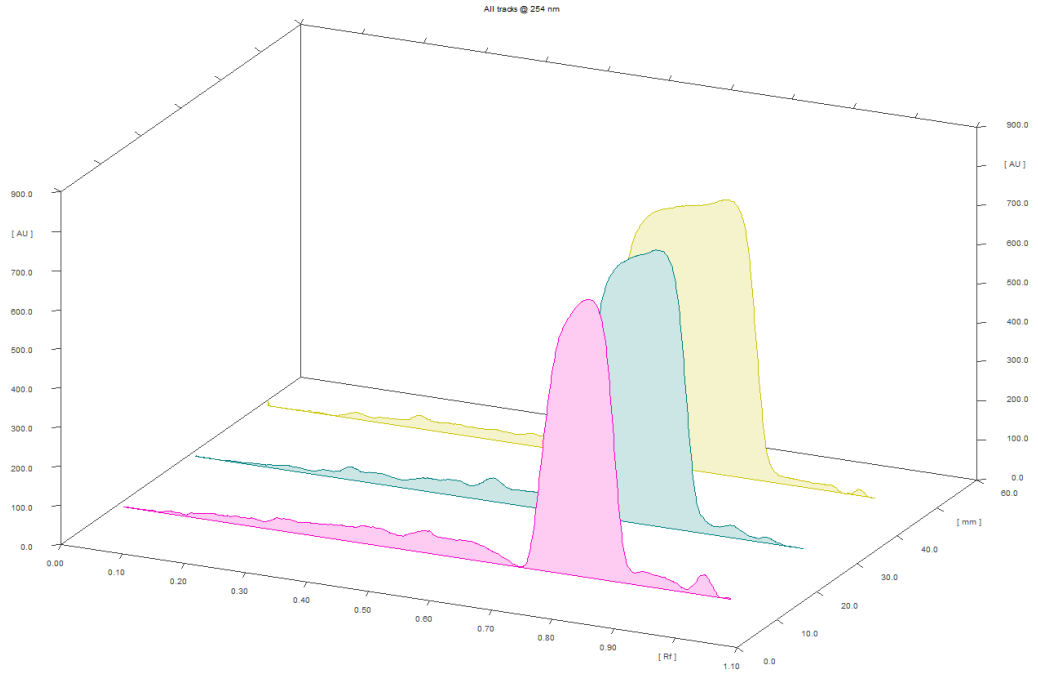
HPTLC Chromatogram @ 254 nm:



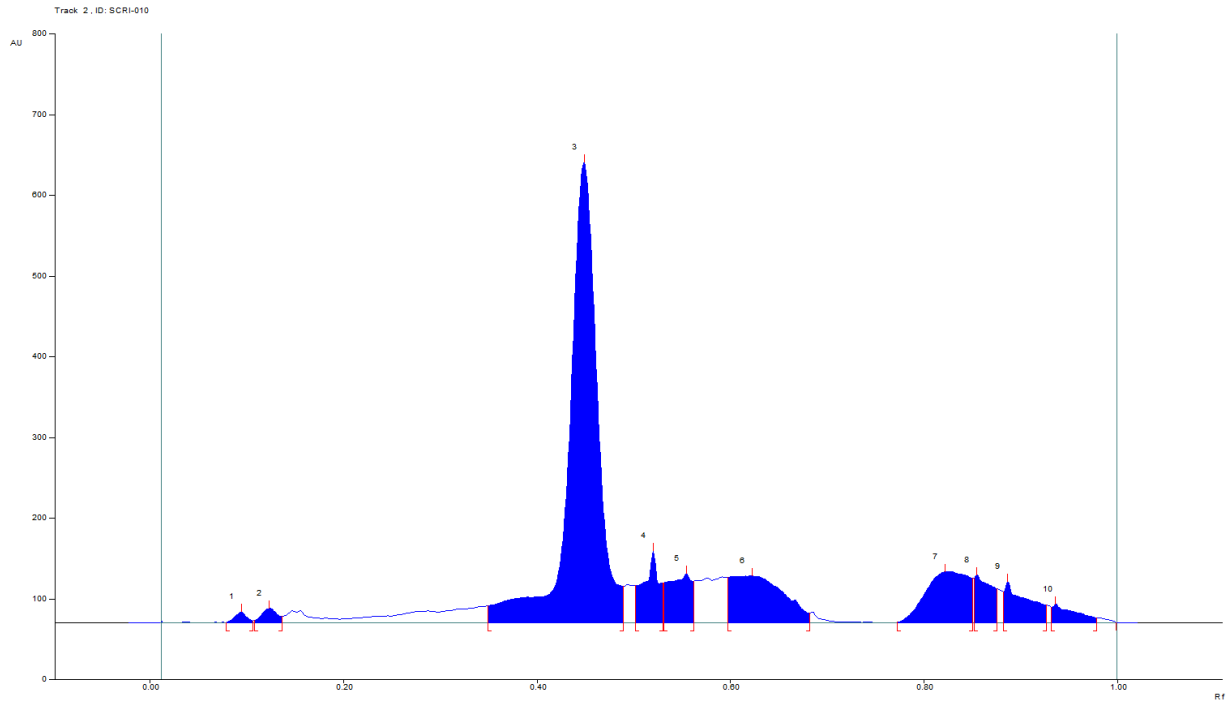
Peak Table @ 254 nm:

Peak	Start Position	Start Height	Max Position	Max Height	Max %	End Position	End Height	Area	Area %
1	0.24 Rf	17.3 AU	0.26 Rf	31.2 AU	3.35 %	0.26 Rf	30.2 AU	565.8 AU	0.71 %
2	0.46 Rf	33.3 AU	0.51 Rf	56.9 AU	6.10 %	0.52 Rf	44.4 AU	2569.6 AU	3.24 %
3	0.56 Rf	44.8 AU	0.58 Rf	46.3 AU	4.97 %	0.65 Rf	0.1 AU	2330.7 AU	2.94 %
4	0.66 Rf	0.2 AU	0.77 Rf	710.2 AU	76.15 %	0.84 Rf	29.4 AU	70936.3 AU	89.37 %
5	0.84 Rf	29.6 AU	0.86 Rf	36.9 AU	3.96 %	0.90 Rf	25.8 AU	1569.8 AU	1.98 %
6	0.93 Rf	7.0 AU	0.96 Rf	51.0 AU	5.47 %	0.98 Rf	0.1 AU	1402.0 AU	1.77 %

3D Chromatogram @ 254 nm:


All tracks @ 254 nm
900.0
[AU]
700.0
600.0
500.0
400.0
300.0
200.0
100.0
0.0
0.00
0.10
0.20
0.30
0.40
0.50
0.60
0.70
0.80
0.90
[Rf]
1.10
0.0
10.0
20.0
30.0
40.0
[mm]
60.0

HPTLC Chromatogram @ 366 nm:

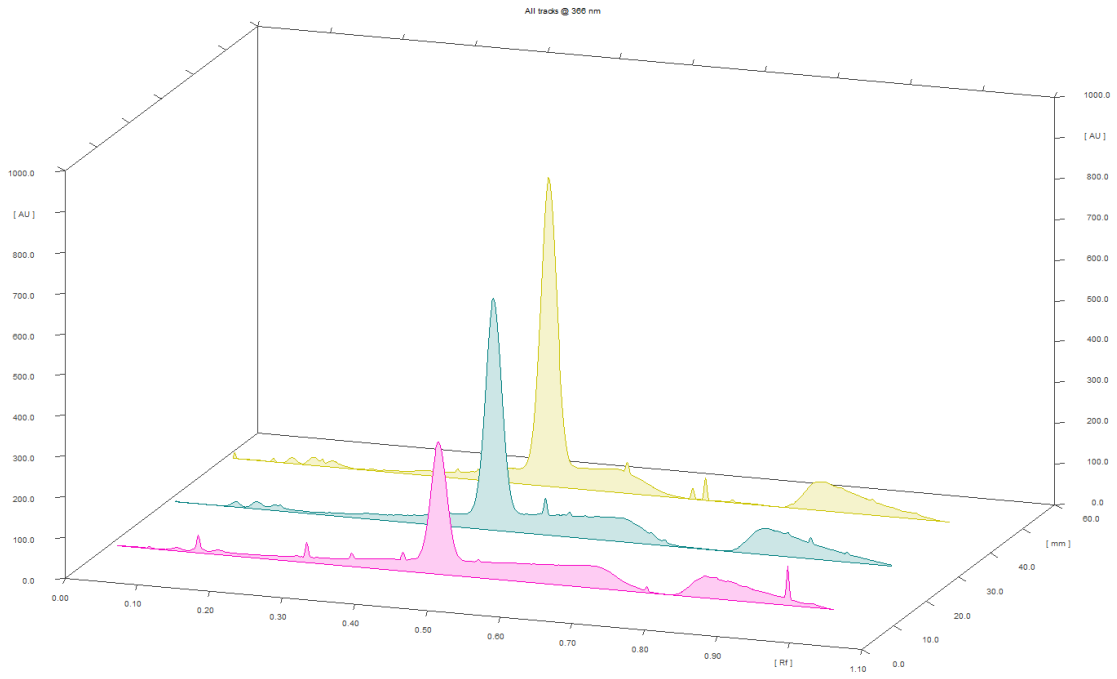


Peak Table @ 366 nm:

Track 2, ID: SCRI-010

Peak	Start Position	Start Height	Max Position	Max Height	Max %	End Position	End Height	Area	Area %
1	0.08 Rf	0.2 AU	0.09 Rf	13.9 AU	1.38 %	0.11 Rf	2.4 AU	174.7 AU	0.57 %
2	0.11 Rf	2.6 AU	0.12 Rf	17.8 AU	1.77 %	0.14 Rf	7.6 AU	302.3 AU	0.99 %
3	0.35 Rf	20.6 AU	0.45 Rf	570.9 AU	56.63 %	0.49 Rf	45.2 AU	17543.5 AU	57.53 %
4	0.50 Rf	46.3 AU	0.52 Rf	89.8 AU	8.91 %	0.53 Rf	49.4 AU	1493.5 AU	4.90 %
5	0.53 Rf	49.7 AU	0.55 Rf	61.0 AU	6.05 %	0.56 Rf	51.7 AU	1540.9 AU	5.05 %
6	0.60 Rf	56.4 AU	0.62 Rf	58.3 AU	5.78 %	0.68 Rf	11.6 AU	3428.1 AU	11.24 %
7	0.77 Rf	0.6 AU	0.82 Rf	63.5 AU	6.30 %	0.85 Rf	54.9 AU	2981.6 AU	9.78 %
8	0.85 Rf	55.2 AU	0.86 Rf	58.9 AU	5.84 %	0.88 Rf	41.9 AU	1140.6 AU	3.74 %
9	0.88 Rf	38.5 AU	0.89 Rf	51.2 AU	5.08 %	0.93 Rf	22.0 AU	1296.0 AU	4.25 %
10	0.93 Rf	19.5 AU	0.94 Rf	22.8 AU	2.26 %	0.98 Rf	5.8 AU	595.0 AU	1.95 %

3D Chromatogram @ 366 nm:



ANNEXURE – IV

TOXICOLOGICAL STUDY

ACUTE ORAL TOXICITY – OECD GUIDELINES - 423

Acute toxicity study was carried out as per OECD guideline (Organization for Economic Co - operation and Development, Guideline-423

Animal: Healthy wistar albino female rat weighing 200–220 gm

Studied carried out at three female rat under fasting condition, signs of toxicity was observed for every one hour for first 24 hours and every day for about 14 days from the beginning of the study.

INTRODUCTION:

The acute toxic class method is a stepwise procedure with the use of 3 animals of a single sex per step. Depending on the mortality and/or the moribund status of the animals, on average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance. Morbid animals or animals obviously in pain or showing signs of severe and enduring distress shall be humanely killed, and are considered in the interpretation of thetest results in the same way as animals that died on test. The method allows for the determination of an LD50 value only when at least two doses result in mortality higher than 0% and lower than 100%.

PRINCIPLE:

It is the principle of the test that based on a stepwise procedure with the use of a minimum number of animals per step, sufficient information is obtained on

the acute toxicity of the test substance to enable its classification. The substance is administered orally to a group of experimental animals at one of the defined doses. The substance is tested using a stepwise procedure, each step using three animals of a single sex. Absence or presence of compound-related mortality of the animals dosed at one step will determine the next step, i.e.; − no further testing is needed − dosing of three additional animals with the same dose − dosing of three additional animals at the next higher or the next lower dose level. The method will enable a judgment with respect to classifying the test substance to one of a series of toxicity classes.

METHODOLOGY

Selection of animal species:

The preferred rodent species is rat, although other rodent species may be used. Healthy young adult animals of commonly used laboratory strain Swiss albino is used . Females should be nulliparous and non-pregnant. Each animal at the commencement of its dosing should be between 8 and 12 weeks old and its weight should fall in an interval within±20 % of the mean weight of the animals.

Housing and feeding conditions:

The temperature in the experimental animal room should be 22ºC (+3ºC). Although the relative humidity should be at least 30% and preferably not exceed 70% other than during room cleaning the aim should be 50-60%. Lighting should be artificial, the sequence being 12 hrs light, 12 hrs dark. For feeding,

conventional laboratory diets may be used with an unlimited supply of drinking water. Animals may be grouped and tagged by dose, but the number of animals per cage must not interfere with clear observations of each animal.

Preparation of animals:

The animals are randomly selected, marked to permit individual identification, and kept in their cages for at least 7 days prior to dosing to allow for acclimatization to the laboratory conditions

Observation done:

Group	Day
Body weight	Normal
Assessments of posture	Normal
Signs of Convulsion Limb paralysis	Absence (-)
Body tone	Normal
Lacrimation	Absence
Salivation	Absence
Change in skin color	No significant colour change
Piloerection	Normal
Defecation	Normal
Sensitivity response	Normal

Locomotion	Normal
Muscle gripness	Normal
Rearing	Mild
Urination	Normal

Dose mg/kg	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**
100	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-

1.*Alertness 2.Aggressiveness 3. Pile erection 4. Grooming 5.Gripping 6. Touch Response 7. Decreased Motor Activity 8.Tremors 9 Convulsions 10. Muscle Spasm 11. Catatonia 12.Musclerelaxant 13.Hypnosis 14.Analgesia15.Lacrimation 16. Exophthalmos 17. Diarrhea 18. Writhing 19 Respiration 20. Mortality*

CONCLUSION:

In the acute toxicity study, the rats were treated with different concentration of RASACHEENEE CHOORANAMfrom the range of 5mg/kg to 100mg/kg which did not produce signs of toxicity, behavioral changes, and mortality in the test groups as compared to the controls when observed during 14 days of the acute toxicity experimental period. These results showed that a single oral dose of the

extract showed no mortality of these rats even under higher dosage levels indicating the high margin of safety of this extract. In acute toxicity test theRASACHEENEE CHOORANAMwas found to be non toxic at the dose level of 100mg/ kg body weight.

Sub-Acute toxicity test

The dose selected for the sub acute toxicity study was 10mg, 20mg/kg of RASACHEENEE CHOORANAM. All the animals were free of intoxicating signs throughout the dosing period of 28 days. No physical changes were observed throughout the dosing period. No mortality was observed during the whole experiment. No abnormal deviations were observed. No significant changes were observed in the values of different parameters studied when compared with controls and values obtained were within normal biological and laboratory limits. The weights of organs recorded did not show any significant differences in the treatment and the control group indicating that RASACHEENEE CHOORANAMwas not toxic to kidney, liver and spleen There was no significant changes were observed in hemoglobin (Hb), red blood cell (RBC), white blood cell (WBC), packed cell volume (PCV), Erythrocyte sedimentation rate (ESR) in all the treated groups as compared to respective control groups. Histopathology studies were carried out on liver, kidney and spleen and recorded.Blood samples for hematological and blood chemical analyses were taken from common carotid artery. All rats were sacrificed after the blood collection. The internal organs and some tissues were observed for gross lesions. All tissues were preserved in 10% neutral buffered formaldehyde solution for histopathological examination.

SUB ACUTE REPORTS

RASACHEENEE CHOORANAM**(10 mg/kg)**

HAEMOTOLOGY

CBC

WBC	:	6500 cells/cumm
Differential Count		
NEUTROPHILLS	:	18%
LYMPHOCYTES	:	76 %
EOSINOPHILS	:	06 %
MONOCYTES	:	02 %
RBC	:	6.5 millions/cumm
HB	:	14.4 gms%
PCV	:	39.6 %
MCV	:	56.5 fL
MCH	:	16.5 pg
MCHC	:	29.3 Grams/dl
PLATELET	:	6.5 Lakhs/cumm

BIOCHEMISTRY

Blood sugar	:	98 mg/dl
BUN	:	36.1 mg/dl
Creatinine	:	0.4 mg/dl
SGOT	:	57 U/L

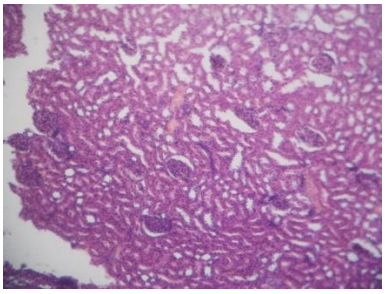
SGPT	:	76 U/L
ALP	:	55 U/L
T.Protein	:	sgrams/dl
Albumin	:	4.5 grams/dl

LIPID PROFILE

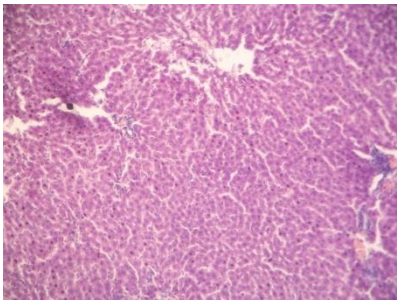
T. Cholesterol	:	131 mg/dl
Triglycerides	:	59 mg/dl
HDL	:	32 mg/dl
LDL	:	67.2 mg/dl
VLDL	:	23.8 mg/dl
Ratio 1(T.CHO/HDL)	:	4.44
Ratio 2(LDL/HDL)	:	2.89

HIISTOPATHOLOGY

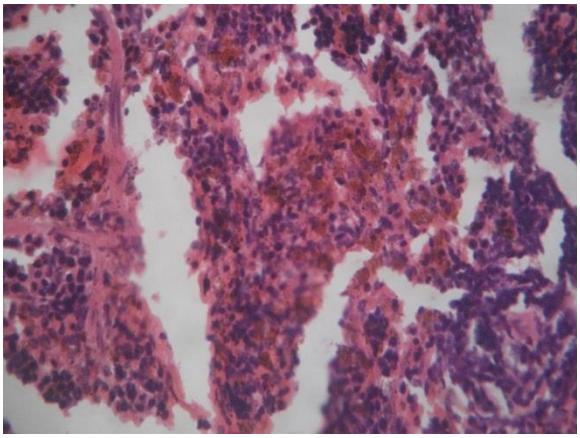
KDNEY



LIVER



SPLEEN



RASACHEENEE CHOORANAM**(20 mg/kg)**

HAEMOTOLOGY

CBC

WBC	:	5,200 cells/cumm
Differential Count		
NEUTROPHILLS	:	10%
LYMPHOCYTES	:	89 %
EOSINOPHILS	:	01 %
MONOCYTES	:	00 %
RBC	:	8.98 millions/cumm
HB	:	16.9 gms%
PCV	:	53.6 %
MCV	:	59.7 Fl
MCH	:	18.8 pg

MCHC	:	31.5 Grams/dl
PLATELET	:	7.20 Lakhs/cumm

BIOCHEMISTRY

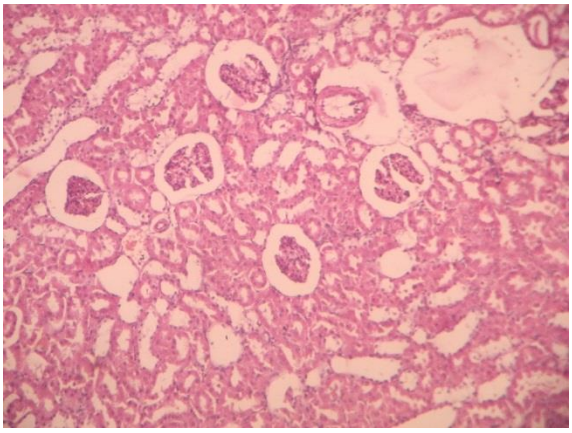
Blood sugar	:	90 mg/dl
BUN	:	52.8 mg/dl
Creatinine	:	0.4 mg/dl
SGOT	:	67 U/L
SGPT	:	44 U/L
ALP	:	45 U/L
T.Protein	:	4.3 grams/dl
Albumin	:	2.2 grams/dl

LIPID PROFILE

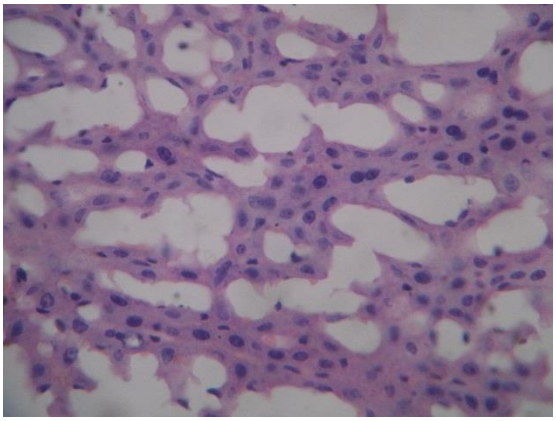
T. Cholesterol	:	112 mg/dl
Triglycerides	:	69 mg/dl
HDL	:	26 mg/dl
LDL	:	72.2 mg/dl
VLDL	:	13.8 mg/dl
Ratio 1(T.CHO/HDL)	:	4.30
Ratio 2(LDL/HDL)	:	2.77

HISTOPATHHOLOGY

KIDNEY



LIVER



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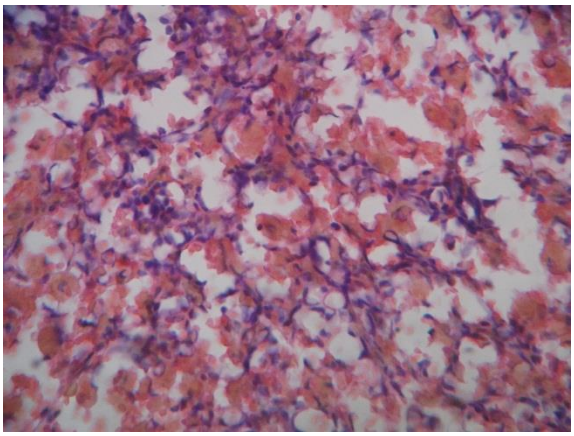

ANNEXURE – V

BIO STATISTICAL ANALYSIS

ANNEXURE - V

BIO STATISTICAL ANALYSIS

CLINICAL PROGNOSIS

Treatment for Venpulli (Vitiligo):

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

S. No	Signs&Symptoms	Before Treatment	After Treatment
		n%	n%
1.	Colour of skin	20(100)	14(70)**
2.	Depigmention of hair	3(15)	2(10)*
3.	Itching	0(0)	0(0)
4.	Erythema	0(0)	0(0)

McNemat test, C.I: 95%, *P<0.05; **P<0.01

Software: spss17 version

Number of cases: 20

Inference:

Since the p value is significant in Colour of skin, Depigmention of hair.So there is significant reducing of Colour of skin, Depigmention of hair among the patients for the treatment of Venpulli (Vitiligo).Hence it is concluded that the treatment was effectiveand **significant.**

VASI SCORE BEFORE AND AFTER TREATMENT

Effect of medicine on VASI Score in human subjects

S.No.	BT VASI Score	AT VASI Score
1	0.1	0.01
2	0.4	0.1
3	0.6	0
4	0.6	0.1
5	3	0.6
6	1.6	0.4
7	0.8	0.2
8	0.8	0.2
9	0.8	0.2
10	0.8	0.1
11	1.2	0.4
12	2.4	0
13	1.8	0.3
14	0.3	0.03
15	1.2	0
16	3.6	0.8
17	1.6	0.4
18	1.6	0.4
19	2.4	0.8
20	2.4	0.8

Software: spss17 version

Variables: VASI Score – before treatment, after treatment

Number of cases: 20

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.734

Before and after treatment mean difference: 1.09 ± 0.78.

P Value (2 tailed): $p<0.001$.

Inference:

Since the P value is highly significant (<0.001). So there is significant reducing of VASI Score among the patients for the treatment of Venpulli (Vitiligo).Hence it is concluded that the treatment was effectiveand **significant.**

HAEMOGLOBIN LEVEL:

Effect of Rasacheenee Chooranam on Hb level (gm/dl)in human subjects

S. no	BeforeTreatment(gms/dl)	AfterTreatment(gms/dl)
1.	10	11
2.	11.5	12
3.	9.4	10.3
4.	9.8	10.4
5.	13	13
6.	11	12.5
7.	13	13
8.	10.2	11.5
9.	10.5	12
10.	9	12
11.	10.4	11.6
12.	9	10.4
13.	9.8	12.4
14.	10.6	11.3
15.	9.9	10.6
16.	10	10.8
17.	11	11.2
18.	9	12
19.	12	13
20.	11	12.3

Software: spss17 version

Variables: Hb level (gm/dl)– before treatment, after treatment

Number of cases: 20

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.684

Before and after treatment mean difference ± SEM: 1.16± 0.86.

P Value (2 tailed): $p<0.001$.

Inference:

Since the P value is highly significant (<0.001), The hypothesis is **not** accepted. So the treatment was significantly improving the Hb level among the patients for the treatment of Venpulli (Vitiligo).

ANNEXURE – VI

CONSENT FORM

ANNEXURE - VI

GOVERNMENT SIDDHA MEDICAL COLLEGE

ARIGNAR ANNA GOVERNMENT HOSPITAL OF INDIAN MEDICINE

CHENNAI – 600 106

CLINICAL STUDY ON **"RASACHEENEE CHOORANAM (INTERNAL) & KARKADAGASINGI PATTRU (EXTERNAL)"** IN THE TREATMENT OF

"VENPULLI" (VITILIGO)

INFORMED CONSENT FORM

"I have read the foregoing information. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction.

I consent voluntarily to participate in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care".

"I have received a copy of the information sheet/consent form".

Date:

Station:

Signature of participant:

Signature of the Guide: Signature of the Investigator:

அரசு சித்த மருத்துவக் கல்லூரி, சென்னை-106

அறிஞர் அண்ணா மருத்துவமனை, சென்னை

வெண்புள்ளிநோய்க்கான சித்த மருந்தின் (*இரசச்சீனி சூரணம்*)உள்மருந்து

கற்கடகசிங்கி (வெளிமருந்து)

பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான தகவல் படிவம்.

ஒப்புதல் படிவம்

ஆய்வாளரால் சான்றளிக்கப்பட்டது

நான் இந்த ஆய்வு குறித்த அனைத்து விபரங்களையும் நோயாளிக்கு புரியும் வகையில் எடுத்துரைத்தேன் என உறுதியளிக்கிறேன்.

தேதி: **கையொப்பம்:**

இடம்: **பெயர் :**

<u>நோயாளியின் ஒப்புதல்</u>

என்னிடம் இந்த மருத்துவ ஆய்வின் காரணத்தையும், மருந்தின் தன்மை மற்றும் மருத்துவ வழிமுறை பற்றியும், தொடர்ந்து எனது உடல் இயக்கத்தை கண்காணிக்கவும், அதனை பாதுகாக்கவும் பயன்படும் மருத்துவ ஆய்வுக்கூட பரிசோதனைகள் பற்றி திருப்தி அளிக்கும் வகையில் ஆய்வு மருத்துவரால் விளக்கிக் கூறப்பட்டது.

நான் இந்த மருத்துவ ஆய்வின் போது, காரணம் எதுவும் கூறாமல், எப்பொழுது வேண்டுமானாலும் இந்த ஆய்விலிருந்து என்னை விடுவித்து கொள்ளும் உரிமையை தெரிந்திருக்கின்றேன். நான் என்னுடைய சுதந்திரமாக தேர்வு செய்யும் உரிமையைக் கொண்டு ***வெண்புள்ளி*** நோய்க்கான ***இரசச்சீனி சூரணம்*** **(*உள்மருந்து), கற்கடகசிங்கி (வெளிமருந்து)*** மருந்தின் பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கு என்னை உட்படுத்த ஒப்புதல் அளிக்கிறேன்.

தேதி : **கையொப்பம் :**

இடம் : **பெயர் :**

தேதி : **சாட்சிக்காரர் கையொப்பம் :**

இடம் : **பெயர் :**

உறவுமுறை :

துறைத்தலைவர் கையொப்பம் : **ஆராய்ச்சியாளர் கையொப்பம்:**

ANNEXURE – VII

CASE SHEET PROFORMA

ANNEXURE - VII

CASE SHEET PROFORMA FOR VENPULLI

GOVT.SIDDHA MEDICAL COLLEGE&HOSPITAL, CHENNAI-106

POST GRADUATE DEPARTMENT BRANCH –I MARUTHUVAM

Duration: 2014-2016

OP No / IP No	:	Occupation	:
Ward No	:	Income	:
Bed No	:	Nationality	:
Name	:	Religion	:
Age	:	D.O.A	:
Sex	:	D.O.D	:
Address	:	Diagnosis	:

1. Complaints and duration :

2. History of present illness :

3. History of past illness :

4. Personal history :

5. Occupational history :

6. Menstrual history :

7. Personal Habits : Veg/non veg/smoker/Alcoholic/Tobacco chewer

8. Family History :

GENERAL EXAMINATION

Patient consciousness :

Body Built :

Nourishment :

Anemia :

Jaundice :

Cyanosis :

Clubbing :

JVP :

Tracheal deviation :

Pedal oedema :

Lymph adenopathy :

VITAL SIGNS

Body Temp :

Pulse :

Respiratory rate :

Blood Pressure :

Heart rate :

SIDDHA ASPECT

NILAM

Kurinji :

Mullai :

Marutham :

Neithal :

Palai :

PARUVA KALAM

Kaar :

Koothir :

Munpani :

Pinpani :

Elavenil :

Muduenil :

YAAKKAI (Udal)

Vatham :

Pitham :

Kabham :

Kalappu :

GUNAM

Saththuvam :

Rajotham :

Thamasam :

PORI/PULANGAL

(SENSORY ORGANS)

Mei –Sensation :

Vaai – Taste :

Kan – Vision :

Mooku - Smell :

Sevi – Hearing :

KANMENTHRIYAM/KANNMA VIDAYAM [MOTOR ORGANS]

Kai- Dhaanam :

Kaal-Kamanam :

Vaai-Vasanam :

Eruvaai- Visarkkam :

Karuvaai-Aanantham :

UTHKAAYA ATHAKAAYAM

Puyam[forearm] :

Sayam[arm] :

Kaal[leg] :

Paaatham[feet] :

UYIR THATHUKKAL

A.VATHAM

Piranan :

Abanan :

Viyanan :

Udanan :

Samanan :

Nagan :

Koorman :

Kirukaran :

Devathathan :

Thananjeyan :

B.PITHAM

Anar pitham :

Ranjaga pitham :

Saathaga pitham :

Pirrasaga pitham :

Alosaga pitham :

C.KAPAM

Avalambagam :

Kilethagam :

Pothagam :

Tharpagam :

Santhigam :

UDALTHAATHUKKAL

Saaram :

Senner :

Oon :

Kozhuppu :

Enbu :

Moolai :

Sukkilam/Suronitham :

ENVAGAI THERVUGAL

1.Naa :

2.Niram :

3.Mozhi :

4.Vizhi :

5.Sparisam :

6.Malam :

7.Moothiram

a)Neer Kuri :

b)Nei Kuri :

8.Naadi :

MALAM

Niram :

Edai :

Erugal :

Elagal :

MOOTHIRAM

1.Neerkuri

Niram :

Manam :

Edai :

Nurai :

Enjal :

2.Neikuri

MODERN ASPECT

Sytemic Examination

Inspection :

Palpation :

Renal angle :

Tenderness : Present/Absent

Radiation :

Percussion :

Auscultation :

Others Systems

Cardio Vascular System :

Respiratory system :

Central nervous system :

CLINICAL SIGN AND SYMPTOMS OF VENPULLI

S.No	Symptoms	Before Treatment	After Treatment			
			10th day	20th day	30th day	40th day
1	Hypopigmentation Area shape					
2	Pruritis					
3	Swelling					
4	Erythema					
5	Depigmentation of hair					
6	Sensation					
7	Scaling					
8	Oozing					

INVESTIGATION

1. BLOOD

TC, DC, ESR
Bleeding time, Clotting time
Blood sugar
Blood urea

2. URINE

Colour
Turbidity
Albumin
Sugar

Deposits

- Epithelial cells
- RBC’s
- Pus cells

3.Thyroid profile
4.Liver function test
5.Renal function test.

TRIAL DRUG:

RASACHEENEE CHOORANAM (INTERNAL)

&

KARKADAGASINGI PATTRU (EXTERNAL)

INTERNAL MEDICINE

Dose: 10 mg bid After food

Adjuvant : palm jaggery

Duration of Treatment: 48 days

Pathiam (Do’s and Don’ts)

Prognosis

Medical Officer Signature: **HOD**

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